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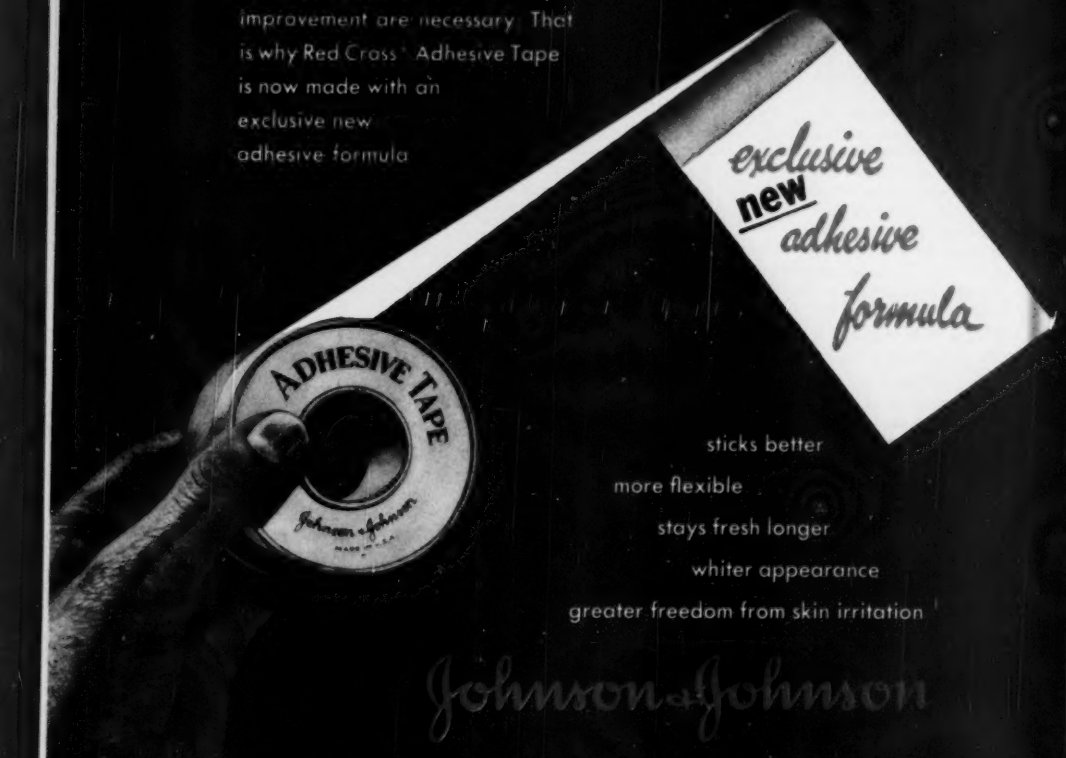

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PSYCHOSOMATIC ASPECTS OF HEARING DEFECTS

Effects of Hearing Loss on Mental Processes

Number 5 of a Series

A defect in hearing initiates emotional and practical problems which profoundly affect the individual's attitude toward himself and society. In turn his handicap influences the way people act toward him. Some aspects of this complex problem are briefly discussed in this time-saving refresher for the busy practitioner.

DEAFNESS OR STUPIDITY?

For centuries deafness and stupidity, if not actual feeble-mindedness, have been linked together in the public mind. This is one of the cruelest misconceptions the uninformed have concerning the deaf. Inability to understand the spoken word was—and is—so great a handicap in social intercourse that it was misinterpreted as mental lack. Even the hard-of-hearing suffered—and still suffer—from this ignorant viewpoint for little differentiation is made in common thought between those who are without the ability to hear the slightest sound and those who have residual hearing in greater or lesser degree.

Perhaps some of the misunderstanding, at least where children are concerned, has arisen because the speech pattern of the hard-of-hearing, especially those with high-tone loss, resembles that of mental retardation.[1] To the untrained person the differences may not be evident, but the trained ear can detect the more level and monotonous tone of the partially deaf. The physician must be alert to avoid the tragic consequences of classifying as mentally retarded the child of normal intelligence.

UNCORRECTED HEARING DEFECTS MAY RESULT IN MENTAL RETARDATION

There is some basis for the belief that there is a connection between mentality and hearing. Best [2] has described it well: "With the deaf, particularly with those born deaf or becoming so in the earliest years of life, to whom is denied the spoken and heard word as a medium of communication with the outside world, the mind is held back; its processes are slowed up; it must perforce remain, as it were, in chains till special means of liberation are brought. The deaf are not only without stimulus of the human voice, but also without the language which is afforded through the avenue of normal and regular speech, and upon which human thought is so largely dependent."

This is applicable to any child whose hearing defect is at all extensive. If no attempt has been made to aid his hearing by mechanical means, or to give him special instruction, he enters school on the level of normal infancy, unaware not only of the meaning of words, but even that objects or qualities have names.[3] No wonder then that the child seems retarded. Indeed, this lack of ability to communicate freely affects adversely his ability to make normal progress at school.[3, 4]

Hearing defects, too long ignored, may actually lead to emotional deficiency resulting in inability to hear. Morkovin [5] cites such an instance in a congenitally deaf boy, who was neglected until late in adolescence. In this case there was residual hearing which, it was found, could be raised to a hearing level by mechanical means. Alas, this aid came too late. Speech was only noise, he could not give meanings to sounds. He continued to communicate in his small circle by means of grunts and signs. "He never grew out of the infantile level intellectually, emotionally, or socially. He was not born feeble-minded, but he actually became so because he failed to learn and develop mentally by deprivation. Because of his isolation he was reduced to an animal state in a civilized community, a human derelict." [5]

One wonders how many children with hearing defects are mistakenly considered feeble-minded, how many with hearing defects *become* feeble-minded, because they lack proper attention in the early formative years of their lives.

DIAGNOSE HEARING DEFECTS EARLY

The problem of the hard-of-hearing child is of the first importance, if these handicapped youngsters are to develop into well-adjusted individuals, self-supporting and useful. Early diagnosis is essential in the case of the very young child, for often those assumed to be totally deaf have islands of hearing which may be stimulated,[6] and the best stimulus is the human voice.[7]

Diagnosis of hearing defects in the very young may be difficult.[6] The common test—a reaction to noise—is not satisfactory for the tactile sense may respond with misleading results.[6]

The problem is one in which the parents should receive the cooperation of teachers, social workers, psychologists as well as physicians.[6] All must be aware that "the sooner deafness is recognized, the better the chance for the individual to grow into a normal human being." [6]

1. Sheridan, Mary D.: The Child's Hearing for Speech. Methuen & Co., Ltd., London, 1948. p. 29.
2. Best, Harry: Deafness and the Deaf in the United States. The Macmillan Co., New York, 1943. Chap. 12.
3. Ewing, I. R. and Ewing, A. W. G.: The Handicap of Deafness. Longmans, Green & Co., London, 1938, Chap. 1.
4. Best, Harry: Idem. Chap. 19.
5. Morkovin, B. V.: Psychological Basis for Auricular Training and Speech Reading of the Acoustically Impaired. In Aural Re-Education: Psychological and Therapeutic Aspects. Hearing Survey Quarterly, 1946, pp. 1-35.
6. Kinkade, J. M.: The Need of a Combined Program of Medical, Educational, Psychological and Vocational Rehabilitation of the Acoustically Impaired. In Aural Re-Education: Psychological and Therapeutic Aspects. Hearing Survey Quarterly, 1946, pp. 36-40.
7. Barnett, H. N.: M. Press. **216**: 174 (Sept. 4) 1946.

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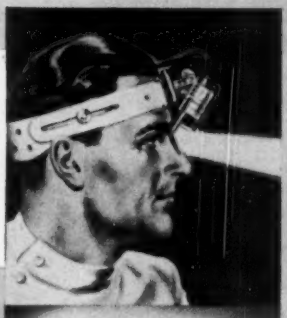

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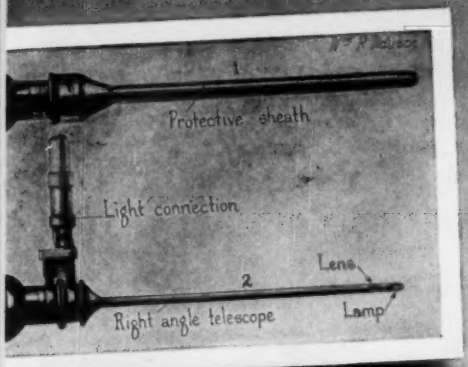

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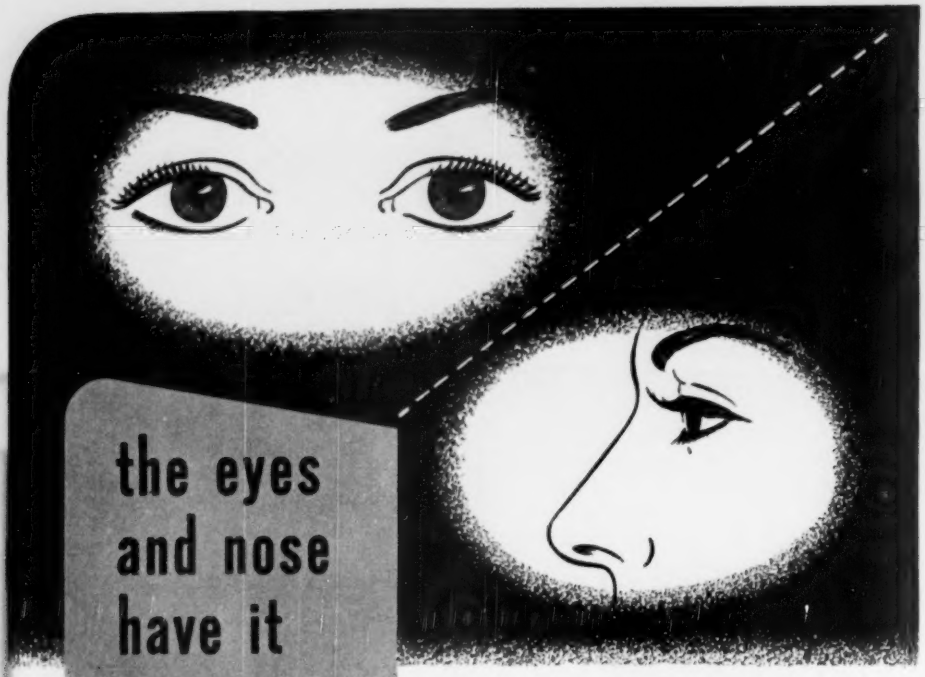

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1. Waldbott, G. L.: Management of Allergic Eczema. Am. Pract. 4:59-63 (Oct.), 1949.

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BONE CONDUCTION THROUGH THE AUDITORY OSSICLE CHAIN

PHILIP G. MacDONALD, M.D.
NEW YORK

IN THE performance of a bone conduction test the bone conduction tip is placed over the mastoid bone. This position of the bone conductor tip gives a theoretic constant. If all mastoid bones conducted sound waves equally well the bone conduction test would give a true estimation of the condition of the cochlea being examined, but the air cell structures of human mastoids vary as to the size, number and distribution of the cells through the bone, and conduction through different types of mastoid bones must vary greatly. The sclerotic mastoid bone cannot have the same function as the normal pneumatic mastoid bone, and sound conduction through the different types of mastoid bones must vary as much in intensity on reaching the cochlea of the ear being tested as the structure varies in the mastoids through which these sounds must pass.

Clinical observations indicate that bone conduction tests may be very misleading, and otologists are now realizing that bone conduction tests do not give a true estimation of the so-called "cochlear reserve of hearing."

Lempert [1] made the following comment on the subject.

> . . . I believe that the significance of the amount of bone conduction hearing for individual sound frequencies found in a particular case with the aid of the audiometer or tuning forks and its relation to the degree of cochlear nerve function is but little understood.
>
> I observed that in a substantial number of cases in which one, two or even all three of the conversational frequencies were heard by bone conduction at a level lower than 30 decibels preoperatively the hearing by air conduction was greatly improved following fenestration and even restored to the practical, serviceable level and in some to normal. . . .
>
> . . . I found that in those cases of clinical otosclerosis in which the involved ear was operated on successfully . . . the hearing for bone-conducted sounds was also improved. The fact that hearing by bone conduction could ever be improved by fenestration of the labyrinth would suggest either that such hearing cannot always be accurately determined with present methods of testing or that bone conduction hearing and cochlear nerve function potential are not synonymous. . . .

1. Lempert, J.: Fenestra Nov-Ovalis with Mobile Stopple, Arch. Otolaryng. **41**:1-41 (Jan.) 1945.

Such experience and observations led me to conclude: first, that the accurate determination of the decibel level of bone conduction hearing is not always possible; second, that the most accurately determined decibel level of bone conduction hearing for individual sound frequencies is not always representative of the degree of cochlear nerve function still present in a deafened ear. . . .

An accurate, infallible test for the determination of the reserve of cochlear nerve function within an ear deafened to air conduction in clinical otosclerosis is yet to be developed.

Monson [2] made the following statement after describing a method of endaural bone audiometry made on the operating table directly from the lateral semicircular canal.

A new method of studying cochlear function by endaural audiometry is here presented. From the results reported by this method it is indicated that there is considerably more cochlear potential than is apparent by the ordinary audiometric bone conduction tests, and the amount of latent cochlear function varies according to the structure of the mastoid bone. These findings have proven helpful in choosing suitable cases for the fenestration operation.

Shambaugh [3] reported cases in which one or more of the speech frequencies by bone conduction were below the 30 decibel level and in which hearing was restored to the serviceable level by the fenestration operation.

MacDonald [4] made the following summary.

The cells of the mastoid process are of great importance in hearing, for they act as sound-absorbing structures, absorbing the sound vibrations that otherwise would build up behind the drum membrane and interfere with and distort its excursions. Undoubtedly, many cases of so-called "frank nerve deafness" diagnosed by tests of bone conduction are not cases of nerve deafness, and the audiograms are misleading. The air cells of the mastoid process may absorb vibrations when a bone conduction test is made and give an erroneous reading, or, again, in cases in which heavy septums are present, may conduct sound waves much better than in the average case and thus suggest that the cochlear reserve is higher than it really is.

The ability of the mastoid air cells to absorb sound is very easily proved in the following experiment.

A. S., who had lost both drum membranes after an attack of scarlet fever in infancy, but who has had dry ears for many years, was chosen for this experiment.

First, roentgenograms of the mastoid processes were obtained (fig. 1). These mastoids are sclerotic, and bone conduction in this type of bone is always greater than in normal pneumatic bone. I should have preferred a subject with pneumatic mastoids, but it is impossible to get pneumatic mastoids in a patient who has had a pathologic condition sufficient to cause destruction of the drum membrane and middle ear contents.

2. Monson, L. P.: A New Method for Determining Cochlear Function by Endaural Audiometry, Tr. Pacific Coast Oto-Ophth. Soc. **29**:228-244, 1948.

3. Shambaugh, G. E.: The Fenestration Operation: An Evaluation of Its Present Status, Surg., Gynec. & Obst. **84**:828-838 (April) 1949.

4. MacDonald, P. G.: Functioning of the Air Cell System of the Mastoid Process in Audition, Arch. Otolaryng. **49**:447-462 (May) 1949.

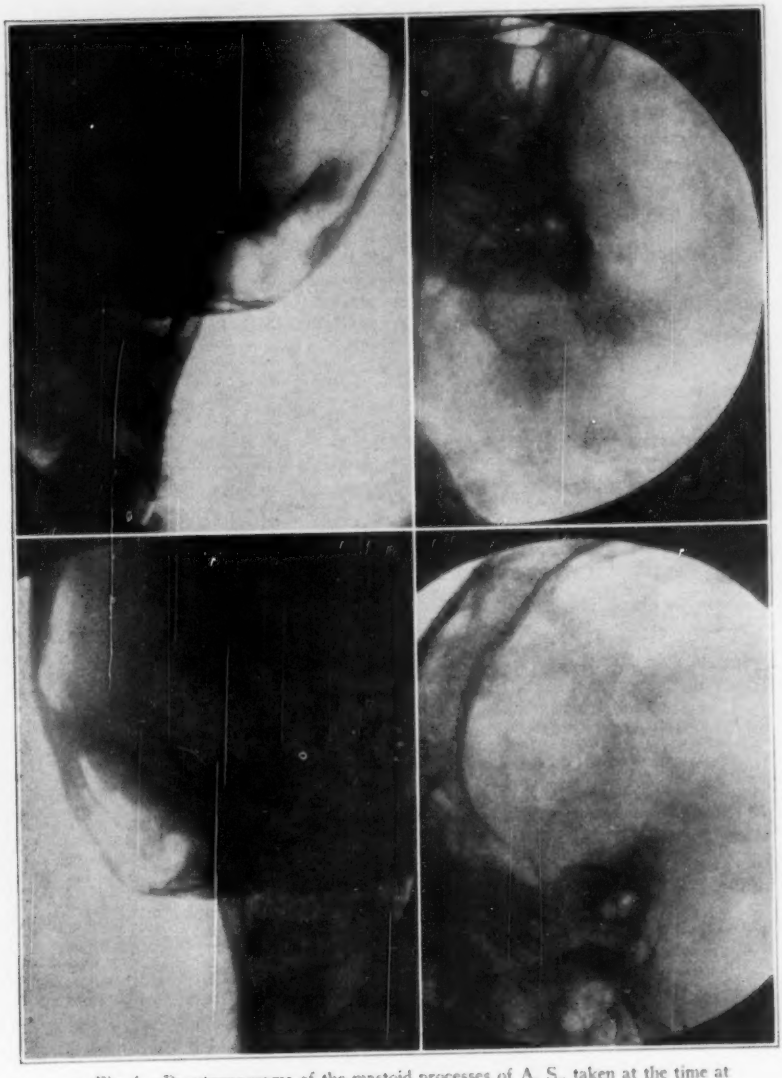

Fig. 1.—Roentgenograms of the mastoid processes of A. S., taken at the time at which they were tested under the conditions described in the legend to figure 2. The roentgenograms at the top are of the left ear; those at the bottom, of the right.

Four audiograms were made for the patient.

1. A bone conduction audiogram was made with the bone conductor tip (fig. 4) placed over the mastoid bone. The screw, *C*, was tightened into the shank, *B*, and used as the contact for the bone conductor against the area of the mastoid bone (see curve *B*, fig. 2).

2. A regular air conduction audiogram was made (see curve *C*, fig. 2).

3. An audiogram was made with the bone conductor held in a malleable frame (see figs. 3, 4, 5 and 6) in front of the ear (fig. 3) and a 20 gage nylon bristle (*E*, fig. 3) passed from the bone conductor to the bone over the prominence of the facial canal where it crosses the middle ear. This nylon bristle has a beaded tip made by heating the end of the bristle in an alcohol flame. The beaded tip was cemented to the prominence of the facial canal with Du Pont cement and the other end of the bristle was engaged in the bone conduction screw adjuster (*C*, fig. 3). Pressure was exerted on the nylon bristle by turning the screw outward. The screw was then adjusted so that the patient felt no pain but only pressure in the ear. (See curve *A*, fig. 2.)

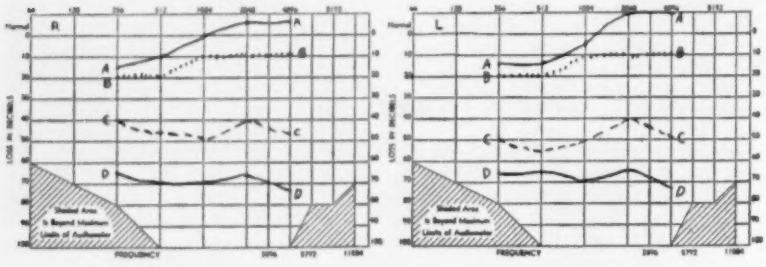

Fig. 2.—Audiograms made for A. S. *R* indicates the right ear and *L*, the left. Curves *A* represent hearing when the nylon bristle conducted the sound vibrations from the bone conduction head to the bone of the prominences of the facial canals where they cross the middle ears. These are corrected curves. The shank and screw (fig. 3) are weights which alter the vibrations of the bone conductor head in the following amounts: 15 decibels at the 256 frequency; 10, at 512; 20, at 1024; 15, at 2048, and 15, at 4096. These figures were therefore subtracted from the frequencies making up audiogram curves *A* and *B*. Curves *B* represent bone conduction hearing with the screw of the shank (fig. 3) of the bone conductor against the area of the mastoid process; curves *C*, regular air conduction hearing, and curves *D*, hearing with the bone conductor head in same position as for curves *A*, but with nylon bristle not inserted.

4. An audiogram was made with the same setup as used in making curve *A* except that in this instance there was no bristle between the adjustment screw and the prominence of the facial canal (curve *D*, fig. 2).

The prominence of the transverse portion of the facial canal as it crosses the middle ear was chosen to be tested in this case, for it was well away from the round and oval windows and so could not directly stimulate the cochlea through one of these windows. The fourth audiogram curve *D* was made to rule out any chance that the patient would hear sounds from the bone conduction head by air conduction, as

in audiogram curve *A*. The threshold of curve *D* is so much higher than that of curve *A* that there can be no chance that this would occur.

It can be noted that the threshold of curve *A* is much less than that of curve *B*. The difference in these thresholds must be due to absorption of sound in the tissue of the mastoid and, to a very slight extent, to the skin and subcutaneous tissue overlying the mastoid cells. The absorption in the skin and subcutaneous tissues is slight, for the bone conduction tip was held firmly against the mastoid area and would thin this tissue out. There may have been a loss of conduction in the nylon bristle that was used in making curve *A*, as the nylon bristle is slightly elastic and may

Fig. 3.—Apparatus in position, with the bristle applied to the facial canal. *A* indicates bone conductor tip held in malleable frame, *G*, and supported on patient's head by a steel spring. *B*, shank attached to bone conductor and carrying adjustable screw, *C*. *C*, screw adjustment to bring pressure on or to release pressure from nylon bristle when apparatus is applied to patient. The center of this screw is drilled, and the nylon bristle (fig. 6) fits into this opening. *D*, prominence over the transverse portion of the facial canal, to which nylon bristle is fastened with Du Pont cement. *E*, nylon bristle, 20 gage, which carries the sound vibrations from the shank of the bone conductor to the prominence of the facial canal. *F*, rubber pads applied to the patient's head as protection from uncomfortable pressure from frame, *G*. *G*, body of malleable frame to hold bone conductor. The legs of this frame are easily bent to help in adjusting instrument to patient's head and are covered with rubber pads as protection from pressure from the apparatus (spring holds frame to patient's head).

have absorbed the sound to a slight extent. I feel, however, that the absorption of sound by the skin and subcutaneous tissue over the mastoid process and the absorption of sound by the nylon bristle are both very

small and can be disregarded in this experiment. All the audiometric curves in this experiment are corrected curves, and this correction will be explained fully in the paper later.

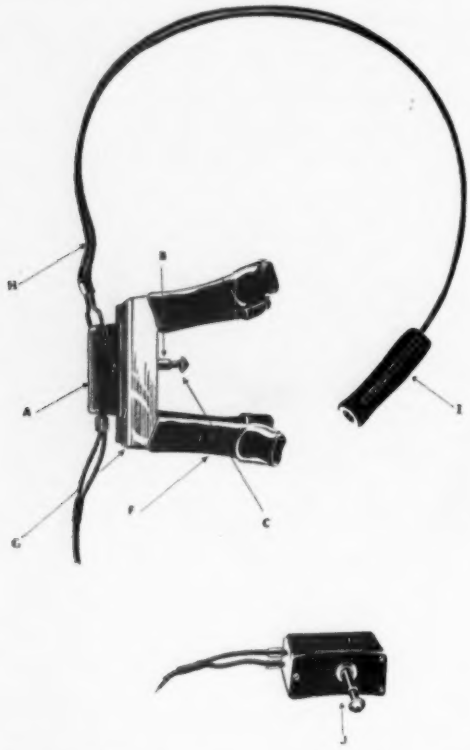

Fig. 4.—Close-up view of the bone conduction tip and the frame to carry the head. *A* indicates bone conduction head, which is held firmly by friction into malleable frame, *G*. The frame and bone conduction head are held to patient's head by the steel spring, *H*. *B*, shank attached to bone conductor head. This shank carries an adjustable screw, *C*. *C*, adjustable screw in shank. This screw can be adjusted inward and outward in the shank and so take up any slack when instrument is adjusted to patient. *F*, legs of frame, which are rubber tipped to prevent any uncomfortable pressure on patient's head. *G*, frame to hold bone conductor head. *H*, steel spring to hold apparatus to patient's head. *I*, rubber pad on end of steel spring. *J*, close-up of head, *A*, to show the shank, *B*, and the screw, *C*, with central drill hole 2 mm. deep and slightly greater bore than the diameter of the 20 gage nylon bristle. The bristle fits into this bore hole and conducts the vibrations to the parts being tested. When the shank and screw are attached to the bone conductor tip, their weight reduces the efficiency of the bone conductor. This reduction in efficiency is corrected in all the curves made with this apparatus.

This experiment is offered as proof that the air cells in the mastoid process act as sound absorbers and that they render the bone conduction test subject to great variation.

This experiment was repeated with several patients, but the results of the bilateral examination of only 1 are shown, for the results were essentially the same in all patients tested.

It can be noted from the roentgenograms that the mastoid processes of A. S. are very sclerotic, with small cells, yet the absorption of sound, as indicated in the audiograms, occurs most in the higher frequencies and least in the lower part of the curves. No anesthetic was used in testing the patients without drum membranes, as they have been wearing

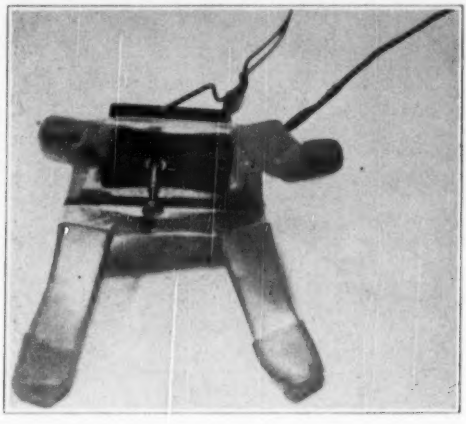

Fig. 5.—Photograph of the apparatus.

so-called artificial ear drums or inserts, and these middle ears were sufficiently insensitive to allow free manipulation without anesthesia. The mastoid air cell systems are not ordinary porous material but definite series of cells connected and lined with a fine, sound-absorbing substance, moist mucous membrane. The architecture of the mastoid process is such that it is really an acoustic labyrinth to disperse and absorb sound waves and as such cannot be considered as being governed by laws which hold when sound is absorbed by ordinary porous substances. The audiograms of A. S. indicate that his mastoids react as ordinary porous material and obey the laws of absorption of sound that is conducted through porous material. In the description of testing pneumatic mastoids, it will be noted that this law does not hold. In

ordinary porous material the attenuation increases with the frequency when sounds are conducted through porous substances.[5]

The question of how much interference was set up by the shank, screw and nylon bristle (figs. 3 and 4) must be considered here. The interference of the shank and screw was determined by making a regular bone conduction test on a patient and then making a second bone conduction test immediately afterward on the same patient but this time the special bone conductor was used (fig. 3), the screw of which was held against the mastoid area, and an audiogram was made. The screw

Fig. 6.—Apparatus on patient.

was tightened into the shank before this test was made. These test audiograms were repeated on a series of patients, and the average increase in the threshold for the different audiometric frequencies when the special bone conductor was used was as follows: 15 decibels at the 256 frequency; 10, at 512; 20, at 1024; 15, at 2048, and 15, at 4096.

For all the curves made with this special bone conductor (fig. 3), these decibels were subtracted from the proper frequencies and the cor-

5. Rettinger, W.: Theory of Sound Transmissions Through Porous and Non-Porous Materials, J. Acoust. Soc. America **8**:172-175 (Jan.) 1937.

rected curves were given. No allowance was made for the nylon bristle in any of the audiograms made with it, since the interference it set up was very slight. This is clearly shown in the case of a patient, G. H., on whom I had performed a successful fenestration on April 25, 1945. The following experiment was made with this patient on Sept. 15, 1948.

A bone conduction test was made with the special bone conductor (fig. 4). The screw was placed against the mastoid area and tightly turned into the shank. (See fig. 7, curve *A*.)

Then a bone conduction test was made from the prominence of the transverse portion of the facial canal. A 20 gage nylon bristle was cemented to the drum membrane over the prominence of the facial canal with Du Pont cement, and an audiogram curve was made (curve *B*, fig. 7).

A curve for bone conduction hearing was made by inserting a 20 gage nylon bristle between the mastoid area and the screw of the special bone conductor. The nylon bristle was fused in an alcohol lamp to form a bead on one end. This end

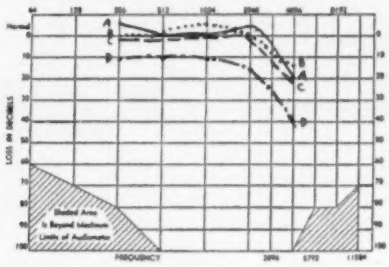

Fig. 7.—Audiograms made for G. H. Curve *A* represents hearing by bone conduction when the special bone conductor (fig. 4) was used over the postfenestrated mastoid area. Screw *C* was applied to mastoid area. Curve *B* represents hearing by bone conduction, also postfenestration, but this time the hearing curve was made from the transverse facial ridge, with use of a nylon bristle and special bone conduction head, as illustrated in figure 3. The drum membrane being present in this case, the nylon bristle was fastened with Du Pont cement to the part of the drum membrane that drapes over the facial ridge. Curve *C* represents hearing with the test made from the mastoid area with the special bone conductor (fig. 4) but with a nylon bristle inserted between screw *C* and the mastoid process. Curves *A*, *B* and *C* were made at the same time, postoperatively, in 1948. Curve *D* represents preoperative bone conduction hearing in 1945. The differences in curves *A*, *B* and *C* are about the same magnitude as the standard deviation in repeated tests and are therefore probably not significant. For this reason curves *A*, *B* and *C* were considered identical in this experimental work (see text).

was fastened to the mastoid area with Du Pont cement and the other end was placed in the screw of the special head. The instrument (fig. 3) was adjusted with respect to pressure on the bristle and the position of the frame; then an audiogram curve (*C*, fig. 7) was made. Curve *D* in the same figure indicates preoperative bone conduction hearing.

On examination of these curves, it can be noted that the preoperative bone conduction curve *D* has a much higher threshold than the other

audiogram curves. The other curves, *A*, *B* and *C*, are very similar, and the differences between them are so slight that for practical purposes they can be considered identical. The distortion on insertion of the bristle is so small, therefore, that I have not taken it into consideration in any of the tests that will be described.

This patient, G. H., had had a very complete mastoidectomy performed at the time of the fenestration, and all the mastoid cells that could be removed were removed. This absence of mastoid cells undoubtedly accounts for the lowering of the threshold of the postoperative bone conduction audiograms in his case.

The preceding experiment suggested a further experiment—that of conduction of the sound waves to the cochlea by bone conduction through the auditory ossicles. This experiment was made with the following technic. This test was performed only with patients with otosclerosis

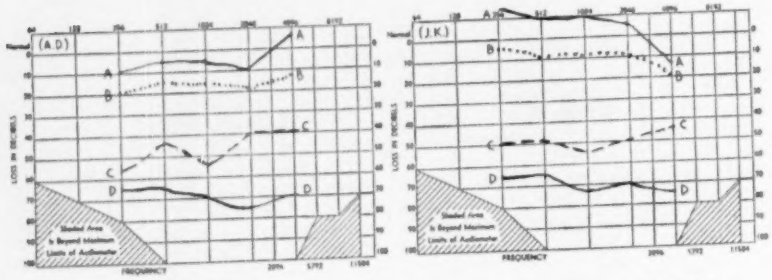

Fig. 8.—Audiograms made for patients, A. D. and J. K., who were to have a fenestration operation. Curves *A* represent hearing when the nylon bristle conducted the sound vibrations from the bone conductor head to the short process of the malleus (fig. 9). These are corrected curves (see legend to fig. 2). Curves *B*, bone conduction hearing. These are also corrected curves. Curves *C*, regular air conduction hearing. Curves *D*, hearing with the bone conductor head in same position as for curve *A*, but with the nylon bristle not inserted. Curve *D* was made to show that there was no chance of hearing the bone conductor in curve *A* by air conduction instead of by bristle and ossicle conduction. These are corrected curves also. For these audiograms two extreme types of mastoid processes were chosen. The mastoid process in A. D. was sclerotic, with small cells, and in J. K., pneumatic with large cells. For J. K., the greater spread between curves *A* and *B* was in the lower tones, and the smaller spread was in the higher tones. For A. D., the smaller spread was in the lower tones and the greater spread was in the higher tones. This situation occurred also when the nylon bristle was attached directly to the bone over the transverse portion of the facial canal of patients who had sclerotic mastoids and no drum membranes.

who were expecting to have a fenestration operation; all the patients tested showed on the Gellé test fixation of the stapes.

The drum membrane of the patient being tested was anesthetized with a solution of cocaine, 12 grains (0.78 Gm.), in aniline, 2 drachms (7.76 Gm.). A pledget of cotton was saturated with the anesthetic and placed against the drum membrane for twenty minutes. The

pledget was then removed and the ear canal wiped well with dry cotton on an applicator. The ear was then thoroughly cleansed with 95 per cent alcohol and ether on cotton applicators in order to remove any oil that might have been left on the drum membrane.

As in the previous experiment, four audiograms were made (fig. 8).

1. A regular air conduction audiogram was made (curve *C*).

2. A bone conduction audiogram was made (curve *B*) with a special bone conductor (fig. 4).

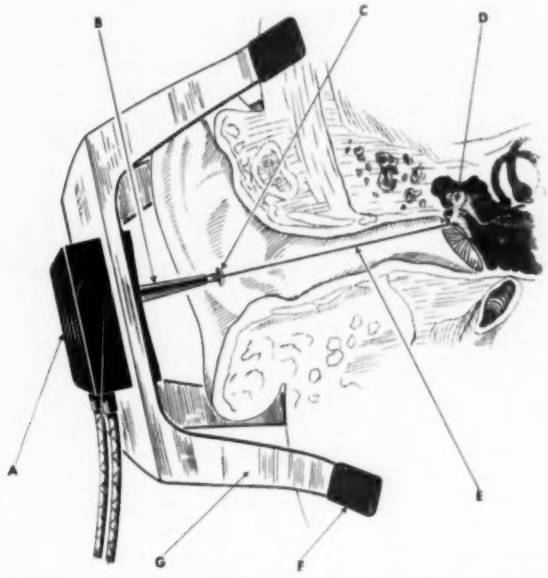

Fig. 9.—Apparatus in position, with the bristle applied to the malleus. *A*, indicates body of bone conductor tip held in malleable frame, *G*. *B*, shank to carry vibrations to nylon bristle, containing adjustable screw, *C*. The center of this screw is drilled to hold the nylon bristle securely (shown in cutaway section). *C*, adjustable screw to bring pressure on or to release pressure from nylon bristle. *D*, malleus, to which beaded tip of nylon bristle is attached. *E*, nylon bristle, 20 gage, which conducts sound vibrations from the bone conductor tip to the short process of the malleus, to which it is held by being attached with Du Pont cement. Just enough cement is used to hold the bristle firmly, for if too much is used, removal of the nylon bristle becomes very painful. *F*, rubber pads for the part of the frame, *G*, that rest on patient's head. *G*, frame that supports the bone conductor tip. This frame is malleable, and its legs can be bent to adjust the shank to the bristle.

3. An audiogram was made (curve *A*) with the bone conductor arranged as shown in figure 9. The nylon bristle in this experiment was attached to the short process of the malleus with Du Pont cement.

Du Pont cement will not stick to the short process of the malleus if oil is present, so the ear, as mentioned previously, is thoroughly cleansed

Results of Ten Consecutive Bone Conduction Tests Through the Ossicle Chain

Case Number	Patient	Age	Frequencies	Decibel Loss: Bone Conduction	Decibel Loss: Ossicle Conduction	Decibel Loss: Difference in Decibels	Ear	Type of Mastoid Process
1	A. D.	24	256	20	10	10	Right	Sclerotic
			512	15	5	10		
			1024	15	5	10		
			2048	20	10	10		
			4096	10	— 5	15		
2	J. K.	21	256	5	—15	20	Right	Pneumatic, with large cells
			512	10	—10	20		
			1024	10	—10	20		
			2048	10	— 5	15		
			4096	20	15	5		
3	A. Z.	38	256	10	—10	20	Left	Pneumatic, with large cells
			512	5	—10	15		
			1024	15	5	10		
			2048	30	20	10		
			4096	35	25	10		
4	K. B.	22	256	20	— 5	25	Left	Sclerotic
			512	15	— 5	20		
			1024	20	—10	30		
			2048	20	— 5	25		
			4096	25	— 5	30		
5	E. R.	46	256	5	— 5	10	Right	Pneumatic, with small cells
			512	15	— 5	20		
			1024	25	10	15		
			2048	25	10	15		
			4096	40	15	25		
6	M. S.	36	256	5	—15	20	Right	Pneumatic, with large cells
			512	5	—10	15		
			1024	15	— 5	20		
			2048	10	0	10		
			4096	20	10	10		
7	M. D.	17	256	5	—15	20	Right	Pneumatic, with large cells
			512	5	—15	20		
			1024	0	—15	15		
			2048	0	—20	20		
			4096	10	— 5	15		
8	H. S.	46	256	25	— 5	30	Right	Pneumatic, with large cells
			512	5	—10	15		
			1024	15	0	15		
			2048	20	5	15		
			4096	25	15	10		
9	J. N.	36	256	20	0	20	Right	Pneumatic, with large cells
			512	25	0	25		
			1024	15	—10	25		
			2048	10	— 5	15		
			4096	5	— 5	10		
10	F. M.	18	256	0	—20	20	Right	Pneumatic, with large cells
			512	5	—10	15		
			1024	15	— 5	20		
			2048	5	—20	25		
			4096	20	5	15		

with 95 per cent alcohol and then ether. The end of the nylon bristle that is attached to the short process is beaded. The bristle is cut in desired lengths, so that little adjustment is needed in applying the bone conductor tip to its distal end (fig. 9). The screw adjustment is turned out until the patient complains of slight pain in the ear being tested. Pressure on the bristle is now reduced until the patient has a sense of

only slight pressure in the ear. The apparatus is now considered set, and an audiogram is made with the double frequencies of 256, 512, 1024, 2048 and 4096 (curve *A*).

4. An audiogram (curve *D*) was made with the setup arranged as shown in figure 9 except that the nylon bristle had not been inserted. This audiogram was made to rule out the possibility of bone conduction sounds being heard by air conduction, as in curve *A*. The threshold of curve *D* is so much higher than that of curve *A* that any such possibility is ruled out. This test has been made with many patients, and the results of consecutive tests with 10 patients are listed in the table.

Examination of the table shows that the ossicle conduction test gives a much lower threshold of hearing than the regular bone conduction test. It will be noted also that the pneumatic mastoid with large cells tends to show a larger spread in the lower tone range, and the pneumatic mastoid with small cells, a larger spread in the high tones.

This test is offered as a check test on bone conduction in cases in which fenestration is being considered. Much more work is necessary on this test before a final opinion can be formed. Many patients now considered inoperable and even patients who are thought to be nerve deafened may be reclassified if retested with the ossicle bone conduction test as described.

SUMMARY

A method for checking bone conduction is offered which can be done in any office and which will help to find marginal cases when fenestration is being considered. Many cases in which diagnosis formerly had been nerve deafness may now be reclassified and may be found to be cases in which bone conduction has been interfered with by absorption in the mastoid air cells to an extent greater than expected. The described test should rule out this interference with the test for bone conduction and, in sclerotic mastoids as well as in pneumatic mastoids, should give the examining physician a clue about what is happening to bone conduction in the mastoid air cells. This test has been performed only with patients with manifest fixation of the stapes. It will be made with other types of patients as well, but as it is a time-consuming test it has not to date been done with any patients other than those selected for fenestration. The results to date, however, warrant a thorough investigation of this test for all patients with deafness, and such a procedure is being planned.

CONCLUSIONS

The test presented here is purely an office procedure but it is time consuming.

The experiments indicate that the mastoid air cells are sound absorbing elements.

This series of tests indicate that the sclerotic mastoid does not absorb sound vibrations as well as the pneumatic mastoid, but it is impossible to differentiate definitely the difference in absorption by the pneumatic mastoid with the large cells from that with the small. If any conclusion could be arrived at, it might be that pneumatic mastoid with the small cells absorbed the high tones better than that with the large cells; but there are not enough cases in this series to warrant a definite statement.

These tests indicate that the mastoid process cannot be compared with ordinary porous material so far as sound absorption is concerned. The mastoid cells are cavities which communicate with each other, forming series of cells, and are lined by sound-absorbing material—mucous membrane.

115 East Sixty-First Street.

FOLD AND CRYPT FORMATION IN THE NASOPHARYNX

An Anatomic and Clinical Study

HANS LION, M.D.
CINCINNATI

THE OBSERVATIONS on a female patient aged 34, who was suffering from spells of a peculiar type of headache, provided the stimulus to begin an exhaustive study of similar cases.

This woman suffered from spells of headache which were of several weeks' duration. These headaches were centered around and behind the right eye. During the time of observation, low grade fever, great fatigue and exhaustion developed and lasted for several weeks. There were no pathologic findings in the upper respiratory tract. The sinuses were normal, but on the roof of the nasopharynx, in the region which connects the upper part of the two tori tubarii, were several deep holes. These holes were embedded in a cobwebbed material in the form of folds and were filled with purulent matter during the spells of headache but were empty in the intervals without headache.

I had observed such hole formation several times in former years and had considered them to represent some kind of embryonic malformation. In order to investigate their origin, their anatomic and clinical function, I started a thorough study of the epipharynx, the structure referred to by T. E. Carmody as "the almost unknown in otolaryngology."

REVIEW OF THE LITERATURE

The literature offers very little material on such formations. (A review of the literature on the subject is presented in a handbook by Denker and Kahler, published by W. Stupka.)

In 1900 Kayser[1] described retronasal synechia and atresia. He stated that there is a great variety in the form of such membrane, ranging from simple little folds that radiate in different directions to complete diaphragms, mostly frontally arranged. He stated the opinion that some of these malformations are hereditary and some acquired. According to Kayser, it is very difficult to obtain specimens for biopsy.

1. Kayser, R.: Verwachsungen der Nase, in Heymann, P.: Handbuch der Laryngology und Rhinologie, Vienna, A. Hölder, 1900, vol. 3, pt. 1.

He referred to Chiari,[2] who, in 1909, found in such biopsy specimens a large number of muscle fibers that were covered on both sides with mucous membrane. In other specimens he found many lymph follicles, glands, blood vessels and round cells, but never muscle fibers. The mucous membrane on one side consisted of columnar epithelium changing into squamous epithelium and on the other side of a simple layer of columnar epithelium without cilia.

Peters described a case in which only scar tissue was revealed in a histologic examination of a 20 year old woman whose adenoids were removed at the age of 11. Gradenigo[5] was of the opinion that the membranous curtains, which stretch from the roof of the nasopharynx toward the tori tubarii, represent involutional processes of the adenoids.

Most of these authors stated the opinion that these folds are found in a much larger percentage in women than in men.

The embryologists Sieur and Rouvillois[4] mentioned these folds in 1912. They considered them to be remnants of a membranous ring which tears and called them primitive velum.

Balla[3] described a case in which a horizontal veil-like membrane had covered almost the entire roof of the nasopharynx and had pulled the right torus tubarius upward and backward. The membrane showed several fissures and several smaller openings. During the night, secretion would collect in the space between the nasopharynx and the membrane and would be expectorated or blown out during the day.

In 1932 Gräff[5] reported on examinations of the nasopharynx in 1000 cases in which autopsies were performed. He mentioned finding stringlike, tapelike and sheetlike adhesions in different sizes between the folds of the adenoids and mucous membrane of the epipharynx. A partial or complete obstruction of one or both of Rosenmüller's fossae is not rare. Gräff held the opinion that to a small degree these changes had its origin in adenoidectomies, but that for the most part they were very likely caused by infections.

Eigler[6] stated that most of the "cysts" are due to retentions in the ducts of the adenoids and the bursa pharyngea. Only in rare cases do

2. Chiari, O.: Ueber angeborene membranöse retronasale Verschlüsse der Choanen, Wien. med. Wchnschr. **59**:561, 1909.

3. Balla, A.: Membrana congenita della faringe nasale, Arch. ital. di otol. **18**:193, 1907.

4. Sieur and Rouvillois: Essai sur le développement embryologique de la cavité naso-pharyngienne, Arch. internat. de laryngol. **33**:466 and 792, 1912.

5. Gräff, S.: Die anatomische Darstellung der Erkrankungen der oberen Luftwege, inbesondere des Epipharynx, Ztschr. f. Hals-, Nasen- u. Ohrenh. **31**:484, 1932.

6. Eigler: Ueber Cystenbildung im Nasenrachenraum, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **147**:364, 1940.

they occur in connection with the craniopharyngeal duct (Theerbrüggen) and on very rare occasions with the branchiogenic ducts (Ginani, Glas).

When the remnants of the adenoids are in the state of involution and become infected, they produce symptoms which resemble very much those symptoms in the condition described as Thornwaldt's bursitis. This disease is an infection of the median recess of the shrunken adenoids. The greatest number of cases of such "abscesses" occur during the ages of 20 to 30. There may be one hole in the epipharynx or several, and sometimes they are 0.5 cc. deep. Most frequently a single opening is found occupying the side of the middle recess of the adenoids. Sometimes three to four openings are found in the median line corresponding to the median cleft of the adenoids. Eigler has actually observed instances in which the condition passed from the stage of abscess to the stage of a cyst and reappeared again as an abscess, all occurring in the median line. These abscesses or crypts were found in adults whose adenoids had never been removed.

The condition occurs shortly after the period when the involution of the adenoid mass has been completed. Eigler was convinced that the condition he has seen is the same as that which Thornwaldt described, but that Thornwaldt erroneously attributed it to a supposedly persistent inflammation in a pharyngeal bursa.

In 1946 Hollender [7] reported findings at autopsy in 140 cases concerning the nasopharynx. Seven cases are mentioned of adhesions in Rosenmüller's fossa. Most of his patients were past middle age, and some were between the ages of 70 and 80.

In 1947 Gidall [8] stated that dysfunction of the lining of the postnasal space, malformation of its contents or repeated inflammatory episodes in this region encourages the production of adhesive bands in the fossae of Rosenmüller as well as in the tubal tonsils and around the eustachian orifice.

ANATOMY AND CLINICAL SYMPTOMS OF NASOPHARYNGEAL FOLD AND CRYPT FORMATION

My survey regarding formations of folds and crypts in the epipharynx was made on 175 unselected patients, who came to our office with various complaints; 103 of the patients, or 59 per cent, were women, and 72 or 41 per cent, were men. All patients were white. We examined 25 patients consecutively, using an epipharynx mirror and a pharyngo-

7. Hollender, A. R.: Some Commonly Unrecognized Diseases of the Nasopharynx: Results of Studies of One Hundred and Forty Autopsy Specimens, South M. J. **40**:248, 1947.

8. Gidall, S. H.: Clinical Significance of Postnasal Space, Eye, Ear, Nose & Throat Monthly **26**:137, 1947.

scope. Those groups of 25 patients were examined during the four seasons of the year in order to have a real cross section of the entire patient material and to exclude seasonal factors. The time of the examinations extended over almost two years. The large number of patients with a variety of fold formations in the epipharynx was greatly surprising.

For statistical purposes it was necessary to arrange the patient material in two general groups. But such an arrangement must be regarded as artificial rather than precise. Another reason for creating two main groups was the probability of the fold formations being produced by different factors.

The first group consisted of patients who showed fold formations above the tori tubarii, either on one or both sides. The folds in this group did not extend toward the center of the nasopharynx.

There are two kinds of folds, sagittal and transverse. The folds extending in a sagittal direction are usually not very pronounced. They

Fig. 1.—Nasopharynx typical of group one. String formation can be seen on the left side, sheet formation on the right. The niches above the tori tubarii are the source of abnormal drainage and inflammation.

are caused by adhesions of the uppermost and anterior part of the torus tubarius with the roof of the nasopharynx. Very often they obstruct the fossa of Rosenmüller, which can normally extend to a depth of 1 inch (2.5 cm.). Of greater clinical importance are the strings and folds extending in a transverse direction. All types of arrangements of these strings and folds can be found, and it can be said that there are hardly two pictures which are similar.

There are strings and bands stretching from the roof of the epipharynx toward the vertex of the torus tubarius or its anterior part (fig. 1). In only 1 case did we find a string descending into the posterior part of the torus. Sheetlike connections between the roof and vertex of the torus tubarius are found, with deep niches between the roof as the anterior border, the sheet as the posterior border and the torus tubarius as the floor. Sometimes I found the arrangement of two

parallel sheets forming two deep niches. These deep niches can be observed as being the source of abnormal secretion or chronic infection with formation and retention of pus.

Group two was comprised of all patients who showed fold formation in the epipharynx beyond a vertical line drawn through the medial border of the torus tubarius. Their folds extended toward the center or were developed there.

This group included 33 patients. In many of these patients one or several folds stretched like curtains from or near the center of the epipharynx toward the upper and medial border of the torus tubarius, leaving between them and the epipharyngeal roof the same kind of niches described for group one. Since the niches in the second group were of greater depth, they were quite often the source of long-lasting or continual postnasal secretion, localized infections, sore throat and

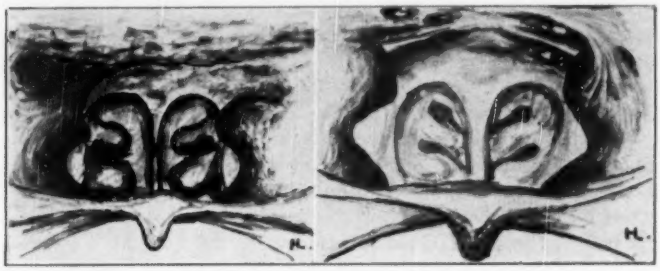

Fig. 2.—Nasopharynx typical of group 2.

other secondary manifestations. It can be assumed that these folds are of utmost importance as a causative factor for long-continued low grade fever of undetermined origin.

Another part of the fold formation exerts a pulling effect in an upward direction on the torus tubarius, thus keeping it wide open.

Some patients with this condition complain that they cannot "click their ear" on the affected side. Usually these patients are easily susceptible to catarrhal conditions, or even infections of the middle ear on the affected side, or else show various degrees of loss of hearing. In air travel they contract aero-otitis easily. The pulling effect on the opening of the eustachian tube is so great in some cases that I find the tube cemented to the roof of the epipharynx, while the opposite side lies sometimes $\frac{1}{4}$ to $\frac{5}{16}$ inch (0.64 to 0.8 cm.) deeper (figure 2, left). This condition very likely causes the kinking or obstruction of

the tube and the sequelae. While watching the closing and opening movement of the orifice of the tube in these cases, one can observe that the anterior and upper border are rendered immovable and that only the posterior border contracts and dilates. In other patients I have seen the tori tubarii pulled toward the center of the epipharynx in a horizontal direction. I have seen strings with their ends attached to the center of the epipharynx and to the torus while the middle part was freely suspended. In 1 case a string extended from one side of the torus to the other, while the medial part floated freely.

The latter half of group two, consisting of 15 patients, presented a most important picture: a large band and sheet formation stretching from the tori tubarii toward the roof and, very often, covering a great part of it. These formations are more or less glued to the anterior and upper surface of the roof but leave cavities between them and the roof. They show one or several small perforations, through which secretion can drain; but in case of nonsufficient drainage, which is especially possible during infection, they reveal various kinds of retention symptoms (fig. 2, right). Patients with this condition are those who have the symptoms described in the beginning of the paper. They are the ones who suffer from a peculiar type of headache which, in my opinion, has never been described before. The headache is one sided, centers around and behind the eye and sometimes radiates toward the forehead or temple. Mucopurulent or purulent secretion can be seen draining through the perforations, but, so it seems, only under pressure exerted from behind. These patients feel ill, are fatigued and listless for weeks; they have long-continued low grade fever; they show loss of weight and very often have a persistent, slight to moderate cough, and their leukocyte count is increased to 10,000 to 15,000. These patients have increasing severity of the symptoms when small or larger parts of adenoid tissue is caught between the network of folds. In case of infection the grip of the strings and sheets surrounding the adenoid tissue prevents expansion and secretion and causes an increase in stress and retention symptoms.

In order to find out statistically whether or not there is any relation between adenoidectomy and fold formation, I have assumed that in all cases in which a tonsillectomy was performed on a patient below 16 years, an adenoidectomy was either performed or at least attempted. As shown in the graph, in group two, in which was presented a much more extended system of fold formation, there was a much higher percentage of tonsillectomies and adenoidectomies than in group one.

Finally, I should like to add a picture which is very well known, namely, the deep crypted atrophic adenoid tissue. Among our 175 patients I found this condition in 10 men, or 14 per cent of all male

patients, and in 14 women, or 13.5 per cent of all female patients. Among these patients during periods of inflammation I found symptoms similar to those previously described.

The picture of tranquility in an interval of freedom from symptoms can be very misleading concerning the diagnosis. On examination, it should be stated therefore what is the prevailing condition of the mucosa or the adenoid tissue. Great mistakes in diagnosis or prognosis can be prevented. Repeated examinations might be necessary in case of a focal infection of unknown cause.

In this connection, the following case report might serve as an illustration.

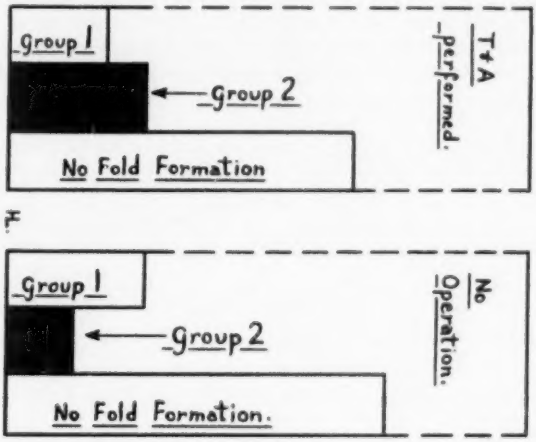

Fig. 3.—Graph comparing percentage of patients with fold formation on whom a tonsillectomy and adenoidectomy had been performed with those patients who had not had the operation.

Two sisters came to the office for a tonsillectomy. M. E., the older sister, was aged 20. In addition to her chronically enlarged tonsils, she exhibited a large clump of adenoid tissue and also some strings with niches, as described for group one. A constant stream of mucopurulent matter was discharged from the niches and adenoids.

J. E., the younger sister, was aged 16. She had the same kind of tonsils as that of her sister, but her nasopharynx seemed entirely free from adenoid tissue or fold formation. Because of the inflammatory condition of M. E.'s tonsils, I scheduled the date of the tonsillectomy three weeks from the date of examination and asked both sisters to come in a few days before the day of the operation. But how greatly surprised I was when I checked the older sister and discovered that her adenoid tissue seemed to have completely disappeared—nor was there any discharge from the niches or any other signs of inflammation. Checking the younger sister, I was

more than startled to find in her nasopharynx, which had not shown any adenoid tissue before, an acutely infected thick clump of such tissue. While performing the tonsillectomy on the older sister, I scraped the epipharynx very thoroughly and was surprised to find that the adenoid tissue was no larger than a medium-sized lima bean.

Although most of the authors cited in the beginning of this treatise stated the opinion that fold formation occurs more frequently in women than in men, I cannot agree with them, according to the statistics shown in the table. I should like to add that the number of patients I have examined is many times greater than theirs.

NATURE AND ORIGIN OF NASOPHARYNGEAL FOLDS

The observations made on a great number of epipharyngeal folds led me to the conclusion that, by far, in the greater number of cases the folds consist of scar tissue and seem to be due to adhesions, which were caused by inflammatory conditions in the nasopharynx during childhood; and that in a smaller number of cases the folds are the result of more

Percentage of Patients with Fold Formation Among 174 Men and Women

	Total Cases	Women		Men	
		Cases	Percentage	Cases	Percentage
Group 1	34	21	20	13	18
Group 2	33	16	15.5	17	23
Totals of Groups 1 and 2	67	37	35.5	30	41

or less faultily performed adenoidectomies. In few cases do they seem to be due to hereditary malformations and in some cases they might be caused by syphilis, tuberculosis and other chronic diseases.

In comparing a sagittal cross section of the nasopharynx of the child and the adult (fig. 4 *A*), it can be seen that the low arch is typical for childhood. The nasopharynx lies in the same level as that of the lower turbinate bone.[9]

Owing to the increasing growth of the face in height and the nasal cavity, the nasopharynx is stretched as high as the middle turbinate bones. While the pharyngeal ostium of the auditory tube rises from the level of the hard palate to the level of the lower turbinate bones, the roof of the epipharynx moves from the level of the lower turbinate to the level of the middle turbinate bone (fig. 4 *B*).

At about the fifth or sixth year, the lymphatic tissue in the nasopharynx has reached its maximum growth. The upper part of Waldeyer's ring, consisting of the pharyngeal tonsil and the two tubal tonsils, represents three large protuberances, which, in many cases, touch

9. Testut, L., and Jacob, O.: Traité d'anatomie topographique avec applications médico-chirurgicales, Paris, O. Doin, 1905, vol. 1.

each other for months or years, or temporarily in an extensive inflammatory condition, on a more or less large surface, as shown schematically in figure 5 *D*. The tubal tonsil consists of lymphatic tissue accumulating in the torus tubarius.

According to Kostanecki, it represents a lateral extension of the pharyngeal tonsil; whereas Gerlach holds the opinion that the lymphatic tissue is a continuation of that tissue of the cartilaginous part of the eustachian tube.

I have observed in children, especially with the nasopharyngoscope, mainly two different kinds of fold formation. The tubal tonsil touches either the roof of the nasopharynx or is in close contact with

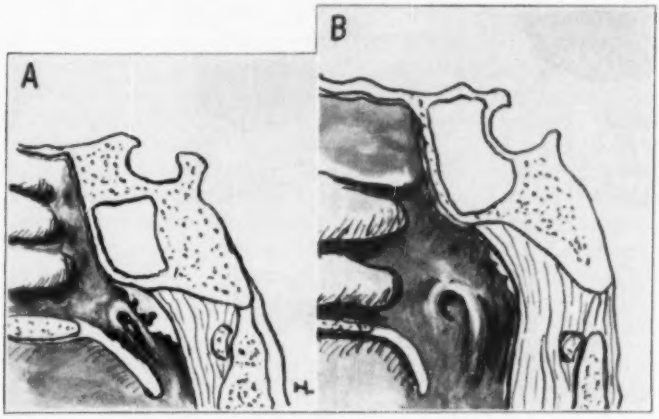

Fig. 4.—Cross section of the nasopharynx (*A*, in the child; *B*, in the adult).

the pharyngeal tonsil. Rarely does it touch a thick clump of lymphoid tissue growing above the torus tubarius.

In my opinion, inflammatory processes may be the cause of epithelial necrosis, which, in turn, produces adhesions of adjacent parts in the nasopharyngeal area. The adhesions will be confined to a small, limited space or to an extended area, depending on the size of the inflamed area, and will result in the formation of strings, bands or sheets or in the gluing together of large surfaces. As soon as the lymphoid tissue in the tubal tonsils and the adenoids starts to shrink, owing to cessation of inflammation, or to contract, owing to involution, the adhesions will be expanded into synechias. Another factor influencing stretching is the increasing growth of the epipharynx, as described previously. This factor must be especially influencing, since not only the tori tubarii but also the adenoids are attached to bony surfaces, while the distance

between the bony surfaces is constantly increasing. The direction of the folds is determined by whether the tori tubarii were contacting the roof of the epipharynx or the adenoids. If the tori tubarii contacted the roof of the epipharynx, the fold formations will take a more or less vertical direction (as described for group one and shown in figure 5, *C*); if they contacted the adenoids, the folds will be directed toward the center (as described for group two and shown in figure 5, *D*). Among those two forms of fold formation, all kinds of variations and transitions are possible.

The second important factor contributing to the formation of folds seems to be caused by more or less faultily performed adenoidectomies.

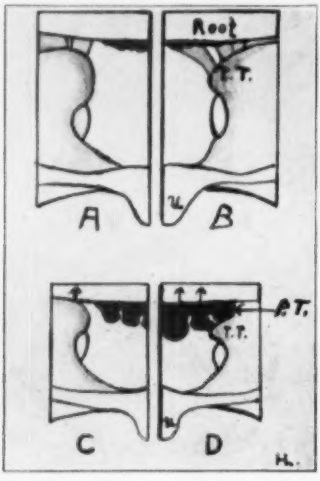

Fig. 5.—Fold formation in the adult (*A* and *B*); in the child (*C* and *D*). *T. T.* indicates the torus tubarius; *U*, the uvula, and *P. T.*, the pharyngeal tonsil.

There is no doubt that some persons produce an excessive amount of scar tissue, even when an operation has been most skillfully performed. If one looks at the statistics in the table, one cannot disregard the fact that the adenoidectomy plays an important part in this group. I have seen conditions among my patients in which a fold coming from the roof stretches, fanlike, into the posterior fold of the torus (fig. 2, right). One receives the impression that the curet has cut deep into this part of the torus, since otherwise it is difficult to understand how an adhesion could form on this place which touches neither the adenoids nor the roof of the epipharynx.

The color of the adhesions and synechias is usually white to grayish white, sometimes light reddish. Their aspect is that of elastic tissue

and their surface is smooth and regular. But in 4 cases I have seen strings, as described by the previously mentioned authors, which have the same color and aspect as adenoid tissue. Their surface is irregular and, in contrast to the tense impression of the synechias, they appear soft and flabby. I cannot fully explain these formations, and I was not able to perform biopsies, but I have no doubts that these strings contain mainly lymphoid tissue and that they might be due to hereditary malformations. They could also be dislocated adenoid tissue.

It might be mentioned here that threads consisting of mucopurulent fluid between the tori and the roof of the epipharynx can be very similar in appearance to adhesions.

Concerning formation of crypts, they are due to dehiscences in the scar tissue or are natural crypts in the atrophic lymphoid tissue of the nasopharynx or, on rare occasions, are due to embryonic malformations.

Because of the copious amount of literature on the subject, I have avoided writing about Thornwaldt's disease. I have seen 1 case of it among 175 patients. But I should like to mention that the headache caused by Thornwaldt's disease and which originates right below the pharyngeal tonsil is a persistent dull aching or throbbing occipital headache localized below the external occipital protuberance; whereas the retention headache, which in my cases, originates on the roof of the epipharynx, consists of pain and pressure around and behind the eye. It is usually one sided and radiates toward the forehead or temple.

THERAPY

The best therapy in cases of fold formation is its prevention. This means that an early, carefully performed adenoidectomy should take place before the adenoids have reached their maximum growth and before severe infections have done excessive damage. When performing an adenoidectomy, cutting through the tori or in any way damaging them should be avoided.

It must be remembered, with special reference to childhood, that although the tonsils might be of normal size, there might exist a hyperplastic picture, with all its destructive power, in the uppermost part of the Waldeyer ring. This process can be detected only by the trained eye. Antibiotics and sulfonamides will help in many cases to prevent severe infections. When folds have developed, it is very difficult in many cases to reduce their damaging power. I have successfully cut single threads by entering into the epipharynx through the nostril, controlling my action with the epipharynx mirror. For this purpose I have used specially made sickle-shaped cutting instruments.

I have treated 2 patients in whom the folds, coming from both tori, were forming a complete arch with the adenoid tissue enclosed

therein. One of the patients was suffering from severe retention symptoms. In both cases I severed the synechias and removed the entire adenoid tissue with a curet. The result was complete cessation of headaches and all other symptoms. In cases in which irremovable adenoid tissue is enclosed in the fold formation and does damage to the ear, or is the origin of an excessive focal infection, radium treatment should be tried.

SUMMARY

A peculiar type of headache which is located around and behind the eye is discussed. It seems to be caused by inflammatory processes within crypts and niches on the roof of the epipharynx.

One hundred and seventy-five unselected patients have been examined and in 67 of them fold formations were found on the roof of the epipharynx.

The anatomy of these folds is presented, and a classification is offered. Etiologic factors responsible for the development of these folds are discussed. The importance of the fold formation is emphasized particularly as being the potential source of frequent focal infections. This aspect of the epipharynx deserves much more attention than it has thus far received. It offers a new approach to its diagnosis and therapy.

523 Doctors Building

THRESHOLD OF FEELING IN THE FENESTRATED EAR

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IN TERMS of atmospheric pressure, the ear is sensitive to a variation of 1/12,000 bar. One-millionth of an atmosphere constitutes a bar. The threshold of hearing is lowest for pitches ranging from 2,000 to 5,000 double vibrations. For the same frequencies, the threshold of feeling is also lowest. An intensity of 100 to 200 bars causes pain in this tonal range. The 16 cycle tone is more felt than heard, while a sound of 20,000 double vibrations requires a pressure of 500 bars to be heard. At the extremely high and extremely low tonal zones, the thresholds of hearing and feeling practically merge.

On the audiogram for the normal ear, the level of minimum audibility is indicated by the "zero line." The threshold of feeling is in the intensity range of 110 to 140 decibels for the frequencies 250 to 5,600 cycles per second. Davis and associates [1] subdivided the sensations of "feeling" into discomfort, tickle and pain. At the Central Institute for the Deaf, in St. Louis, the determinations indicated in the table were obtained by Davis and his associates [1] on 46 normal and 46 hard of hearing ears.

In clinical practice with untrained subjects it is difficult to get descriptions of the distinctive sensations—discomfort, tickle, pain. The patient merely flings the receiver away from the ear with the statement that "it is unbearable." In our clinic the audiometers in use are calibrated to the limit of 100 decibel intensity for frequencies 512 to 4,096. It is only occasionally that the complaint of discomfort from an intensity of 100 decibels for the middle frequencies is registered on routine audiometric examination. We came to recognize the amount of sound pressure lying on the equal loudness contour curve with a value of 100 decibels for frequency 1,024 (Fletcher and Munson [2]) as being tolerable

From the Department of Otorhinology, Temple University School of Medicine.

1. Davis, H.; Hudgins, C. V.; Marquis, R. J.; Nichols, R. H., Jr.; Peterson, G. E.; Ross, D. A., and Stevens, S. S.: Laryngoscope **56**:85 and 135, 1946.

2. Fletcher, H., and Munson, W. A.: J. Acoust. Soc. Am. **5**:82, 1933.

to most ears. The specific intensities are 70 decibels for the 128 frequency; 80 for the 256; 100 for the 512 to 4,096, and 80 for the 8,192. While a few ears with perceptive deafness may be genuinely "tender" to loud sounds, a psychogenic component is responsible for the complaint of discomfort at the 100 decibel level in some instances. It is the psychic makeup of the individual person rather than his ears that is responsible for the reaction.

During the past ten years we have had the opportunity of studying more than 50 patients who had undergone the fenestration operation. Invariably, an intensity of 100 decibels for the middle frequencies could not be tolerated by the fenestrated ear. In many cases discomfort was experienced in the ear that had been operated on with the medium tones

Determinations of Threshold of Feeling in 46 Normal and 46 Hard of Hearing Ears by Decibels

	Discomfort		Tickle		Pain	
	Initial	Final	Initial	Final	Initial	Final
Normal Ears						
Pure tone	110	120	133	142	140	142
Speech	117	130	129	135	138	136
Hard of Hearing Ears						
Pure tone	119	129	129	141	130	141
Speech	121	130	129	134	135	137

at the 80 decibel level; whereas in the ear not operated on tolerance for the same frequencies at an intensity of 100 decibels was usually present. As this phenomenon was present in every case, a psychic element as the predominating factor could be ruled out. The difference between the two ears was striking enough in every instance to justify the conclusion that the altered physiology in the fenestrated ear brings about a marked lowering of the threshold of feeling.

REPRESENTATIVE CASES

Case 1.—J. P., a white woman aged 32, married, had clinical otosclerosis. The hearing loss was practically identical in both ears. On July 18, 1949 the fenestration operation was performed on the right ear. The patient has been socially rehabilitated since the operation, and her audiogram shows a 25 to 30 decibel improvement for the speech frequencies. All frequencies are tolerated at the highest intensity of the audiometer on the ear not operated on. The threshold of pain is reached in the fenestrated ear at 80 decibels for 256 double vibrations; 100 for 512; 95 for 1024; 85 for 2048 and 4096, and 80 for 8192.

Note.—By the equal loudness contours, the most significant lowering of the threshold of feeling is shown in frequencies 2048 and 4096.

CASE 2.—M. F., a white woman aged 24, single, for whom the diagnosis of clinical otosclerosis had been established, had undergone the fenestration operation on the right ear on Feb. 25, 1947. Her preoperative hearing defect was practically similar in both ears. The thresholds of hearing and discomfort, as of Nov. 15, 1949, are shown in chart 2.

NOTE.—In spite of the evident closure of the fenestra, the right ear is exceedingly "tender." The lowering of the threshold of feeling is most pronounced for frequencies 2048 and 4096.

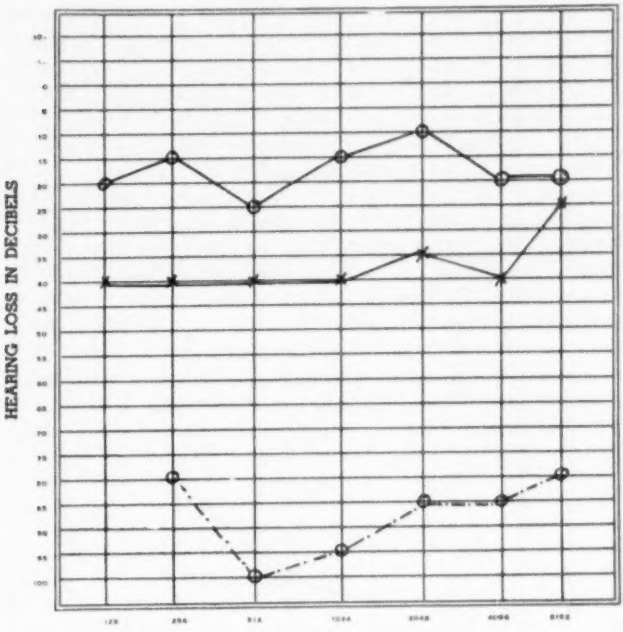

Chart 1.—Audiogram for J. P. As in charts 2 and 3, the circle indicates the right ear, the cross, the left; the unbroken line indicates threshold of hearing and the broken line, threshold of "feeling," in the fenestrated ear.

CASE 3.—M. P., a white woman aged 52, married, who had had clinical otosclerosis, had undergone the fenestration operation on the left ear in 1940. She was socially rehabilitated for about six to seven years. Since 1947 her deafness has been of a handicapping degree, and since 1948 she has been "considering" obtaining a hearing aid. Her hearing and "feeling" thresholds, as of Nov. 10, 1949, are presented in chart 3.

NOTE.—There is a decided lowering of the threshold of feeling for frequencies 2048 and 4096. The deterioration of acuity for high tones in the fenestrated ear is a noteworthy feature.

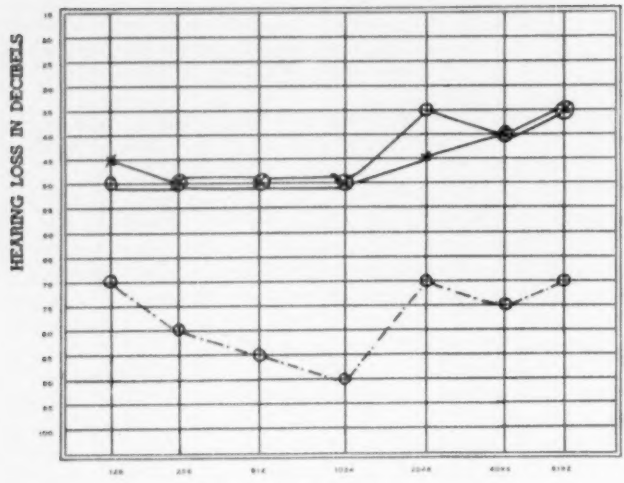

Chart 2.—Audiogram for M. F.

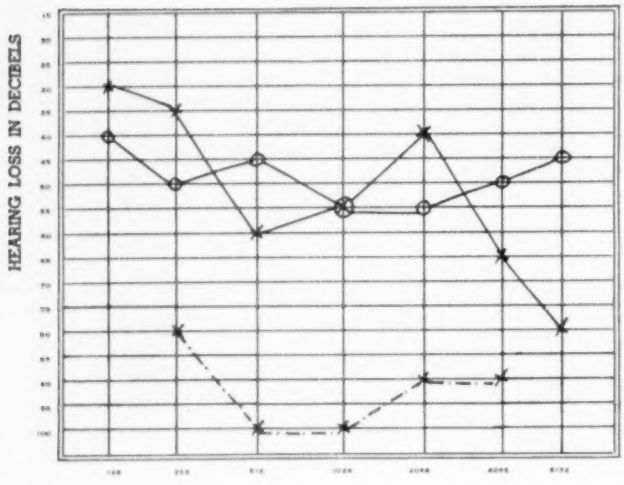

Chart 3.—Audiogram for M. D.

COMMENT

A new physiology of the ear is created by the fenestration operation. In the old physiology, the transference of the sound waves from a medium of low density—air—into a medium of high resistance—perilymph—was accomplished through the intensification of the vibrations by the leverage action of the ossicles. Another factor was the greater effective pressure at the membrane of the oval window, due to the membrane being one-twentieth the size of the membrana tympani. Still another factor was the increase in the acuity for the high tones brought about by the contraction of the stapedius and tensor tympani muscles. The fenestration physiology provides for an exposure of the sensory epithelium to the direct ingress of sound vibrations. Nature provided the intra-aural muscles for the protection of the inner ear from injury incident to loud sounds, according to most physiologists. Wiggers[3] has shown that the contraction of the tensor tympani and stapedius muscles reduces the vibration of low frequencies. In the fenestration physiology, no provision has been made to safeguard the cochlea against acoustic trauma. We found that the threshold of feeling was lowered in the fenestrated ear, and that the lowering of the threshold was more pronounced in the frequency zone of 2,048 to 4,096. Acoustic trauma may well account for the deterioration of acuity for high tones in the fenestrated ear seen months or years after the operation.

A point of practical importance may be brought home. It concerns the person with otosclerosis who has undergone the fenestration operation and who is employed in a noisy occupation. He is to be advised to wear an "ear warden" or other suitable ear protector when exposed to loud sounds.

1923 Spruce Street.

1915 Spruce Street.

3. Wiggers, H. L.: Am. J. Physiol. **120**:771, 1937

FORCED CIRCLING MOVEMENTS (ADVERSIVE SYNDROME)

Correction with Dimenhydrinate ("Dramamine")

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AND
H. E. HIMWICH, M.D., Ph.D.
MEDICAL DVISION, ARMY CHEMICAL CENTER, MD

EXPERIMENTAL production of forced circling movements is well known, these movements having been elicited by means of anatomic lesions in numerous areas of the central nervous system. Muskens [1] produced such lesions by sectioning fibers of the posterior longitudinal fasciculus. Kennard and Ectors [2] reported forced circular movements after ablating area 8 of one frontal lobe. Mussen,[3] on the other hand, obtained this effect by stimulating the red nucleus. Chemical alteration also may evoke compulsive behavior. Goldin and associates [4] noted circling movements with the use of nitrogen mustards. Hill and associates [5] observed similar behavioral changes in rats placed on diets deficient in manganese. However, the production of compulsory turning by a

From the Medical Division, Army Chemical Center, Maryland.

Read before the Section on Otolaryngology, College of Physicians, Philadelphia, on Jan. 18, 1950.

Thesis accepted by the faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science [M. Sc. (Med.)] for graduate work in Otolaryngology.

1. Muskens, L. J., cited by Brock, S.: The Basis of Clinical Neurology: The Anatomy and Physiology of the Nervous System in Their Application to Clinical Neurology, Baltimore, William Wood & Company, 1937, chap. 10.

2. Kennard, M. A., and Ectors, L.: Forced Circling in Monkeys Following Lesions of the Frontal Lobes, J. Neurophysiol. **1**:45-54, 1938.

3. Mussen, A. T.: Cerebellum and Red Nucleus: A Preliminary Report on a New Method of Physiologic Investigation, Arch. Neurol. & Psychiat. **31**:110-126 (Jan.) 1934.

4. Goldin, A.; Noe, H. A.; Landing, B. H.; Shapiro, D. M., and Goldberg, B.: A Neurological Syndrome Induced by Administration of Some Chlorinated Tertiary Amines, J. Pharmacol. & Exper. Therap. **94**:249-261, 1948.

5. Hill, R. M.; Holtkamp, D. E.; Buchanan, A. R., and Rutledge, E. K.: Ataxia and Loss of Equilibrium in Manganese Deficient Rats, Federation Proc. **8**:206, 1949.

biochemical lesion was first observed by Freedman and Himwich,[6] who elicited this response, called the adversive syndrome, by the intracarotid injection of di-isopropyl fluorophosphate, a drug which destroys cholinesterase and permits the accumulation of acetylcholine. They found that atropine or scopolamine hydrobromide corrected the abnormality. Further elaboration on this syndrome and additional data indicating its central origin have been observed by Essig and associates.[7]

In an attempt to find a substance which might correct this turning syndrome, it was decided to investigate the action of dimenhydrinate ("dramamine"). This compound consists of diphenhydramine hydrochloride, commonly called benadryl hydrochloride,[8] and 8-chlorotheophylline and has some atropine-like activity.[8] Dimenhydrinate is used in the treatment of vestibular dysfunction associated with motion sickness,[9] which suggested its use for this study.

It is the purpose of this paper to define the locus of action of di-isopropyl fluorophosphate in the turning syndrome by further investigation of the labyrinthine end organ. Unilaterally labyrinthectomized rabbits [7] who had received injections with di-isopropyl fluorophosphate on the side that had been operated on failed to show circling behavior. The result of sectioning the eighth nerve in rabbits and cats indicated that the vestibular portion of the inner ear may have a secondary role in the production of turning behavior.

METHOD AND RESULTS

The forced circling movements (adversive syndrome) were induced by the following method: With local anesthesia the common carotid artery of a rabbit was isolated operatively and 0.1 mg. of di-isoproply fluorophosphate per kilogram of body weight was injected into it. Additional doses were used if necessary to bring on the syndrome. Within a minute or two there appeared a decided constriction of the pupil on the side that had been operated on. This constriction occurred in all the animals used and confirmed the observations of other investigators.[10] Within a few moments the head appeared to be compulsively turned away from the side into which the injection had been made and the chin tended to point

6. Freedman, A. M., and Himwich, H. E.: DFP: Site of Injection and Variation in Response, Am. J. Physiol. **156**:125-128, 1949.

7. (*a*) Essig, C. F.; Hampson, J. L., and Himwich, H. E.: Biochemically Induced Circling Behavior, J. Nerv. & Ment. Dis., to be published. (*b*) Essig, C. F.; Hampson, J. L.; McCauley, A., and Himwich, H. E.: An Experimental Analysis of Biochemically Induced Circling Behavior, in preparation.

8. Hunter, R. B., and Dunlop, D. M.: A Review of Antihistamine Drugs, Quart. J. Med. **68**:271-290, 1948.

9. Gay, L. N., and Carliner, P. E.: The Prevention and Treatment of Motion Sickness, Bull. Johns Hopkins Hosp. **84**:470-490, 1949.

10. Freedman and Himwich.[6] Footnote 7.

slightly upward, as though the animal was looking over its shoulder. When the animal was released, it became apparent at once that compulsive turning movements had completely replaced forward progression. The animal was compelled to pivot on its hindlegs or to circle on all four legs always in the direction away from the side into which the injection had been made. During this period of active turning, the animal was able to maintain its balance and could right itself normally. The visual system did not appear to be involved, since blindfolding did not alter the course of the syndrome. Head and ocular nystagmus were present, showing a fast and slow component. If the quantity of di-isopropyl fluorophosphate was increased a generalized convulsive seizure was brought on.

Seven different kinds of experiments were performed in an attempt to test the role played by the inner ear in this syndrome:

1. First, we decided to try experimental application of di-isopropyl fluorophosphate to the inner ear. The fact that a solution placed in the middle ear can influence the activity of the vestibular part of the membranous labyrinth has been demonstrated by Spiegel.[11] When a 5 per cent solution of cocaine hydrochloride was injected through the drum membrane into the middle ear of each of the cats used in this experiment, the animal began to turn and circle in the exact manner as with the adversive syndrome, but opposite in direction. The turning was toward the ear into which the injection had been made, as was the slow component of the nystagmus. These findings corroborate the work of Spiegel and prove further that it is possible to paralyze and affect the inner ear via the round window of the middle ear. It is interesting to observe that cocaine injected into the middle ear also produced miosis and paralysis of the nictitating membrane on the side into which the injection had been made and thus reproduced the same ocular effects as injections of di-isopropyl fluorophosphate did.

2. Fortified with the results of this transtympanic injection, we decided to inject 0.5 cc. of di-isopropyl fluorophosphate (1 mg. per cubic centimeter) into the right middle ear of each of 2 cats. Neither animal showed turning, pupillary change or nystagmus. It is well known that di-isopropyl fluorophosphate and allied compounds are both water-soluble and lipid-soluble and should be easily absorbed through the round window. This fact is in agreement with Spiegel's experiment[12] with di-isopropyl fluorophosphate and other parasympathomimetic drugs.

11. Spiegel, E. A.: Production of Labyrinthine Paralysis by Application of Local Anesthetics to External and Middle Ear, Proc. Soc. Exper. Biol. & Med. **56**:137-139, 1944.

12. Spiegel, E. A., in discussion on Essig, C. F.; Hampson, J. L.; Bales, P. D., and Himwich, H. E.: Biochemically Induced Forced Circling Behavior, Tr. Am. Neurol. A. **74**:154, 1949.

3. In order to eliminate the chance possibility that the substance might not come into contact with the round window, the bullous mastoid air cell was opened and the round window plainly visualized. A small pledget saturated with di-isopropyl fluorophosphate was inserted into the niche of the round window in intimate contact with its membrane. The incision was closed, and the animals were observed for several hours. At no time was any unusual movement observed in the 2 cats so treated.

4. To press still further the point of locus of action, a small, fine, curved glass structure was made and, under magnification, inserted carefully through the round window. A small amount (0.02 cc.) of di-isopropyl fluorophosphate was injected slowly and permitted to wash back and forth by capillarity. No apparent effect was noted to indicate activity of the drug in either of the 2 cats used. No evidence of loss of perilymph was observed.

5. After the intracarotid injection of di-isopropyl fluorophosphate in 8 rabbits, each animal showed the classic adversive syndrome. The animal then was given 5 mg. of dinenhydrinate per kilogram of body weight intravenously. This drug appeared to correct completely the turning effect of the syndrome, and the animal was able to walk straight, or to the left and right, with ease.

6. An identical corrective action was observed after the intravenous administration of 5 mg. of diphenhydramine hydrochloride per kilogram of body weight in another group of 4 rabbits who had previously received injections of di-isopropyl fluorophosphate.

7. When 4 rabbits were given 10 mg. of dinenhydrinate per kilogram of body weight prior to the intracarotid injection of 0.2 mg. of di-isopropyl fluorophosphate per kilogram of body weight, no turning effort appeared to be made. Each animal was able to progress in a straight line. A less degree of miosis was observed on the side into which the injection had been made with di-isopropyl fluorophosphate. This occurrence was probably due to the atropine-like action of dinenhydrinate producing a mydriasis, which partially neutralized the miotic effect of di-isopropyl fluorophosphate.

COMMENT

This series of experiments shows that the compulsive turning syndrome produced by the intracarotid injection of di-isopropyl fluorophosphate is not mediated via the inner ear in the cat, though in different circumstances it can be evoked from peripheral vestibular receptors, as seen in the cocainized animal. The action of di-isopropyl fluorophosphate is to destroy the enzyme cholinesterase. This enzyme is found in many tissues of the body, and its specific activity is to facilitate the hydrolysis of acetylcholine. It would seem, therefore, that the intracarotid injection

of di-isopropyl fluorophosphate produced its action either (1) by perpetuating the activity of acetylcholine in the peripheral end organ of the labyrinth or (2) by exerting a central activity through stimulation of cerebral structures connected with vestibular function. Evidence for the participation of striatal structures in vestibular activity indicates that these structures may take part in the forced circling behavior.[13]

If the di-isopropyl fluorophosphate had been able to affect the neuroepithelial elements of the labyrinth with the entrance of this drug into the inner ear, the animal should have shown some type of turning motion. But the animals showed no activity after application of this drug to the inner ear structures. It is logical, therefore, to conceive that the neuroepithelium is not cholinergic and that the application of drugs perpetuating the action of acetylcholine is without effect. If this is true, then the intracarotid injection of di-isopropyl fluorophosphate certainly should not cause turning by its action on the inner ear.

We have shown by the fifth and the seventh experiment that dinenhydrinate is capable not only of curing the central vestibular effects of di-isopropyl fluorophosphate, but also of preventing them, though we arbitrarily chose a dose twice as great to prevent turning as that used to correct it. It has been interesting to observe that diphenhydramine hydrochloride, one of the components of dinenhydrinate, produced many effects similar to those of dinenhydrinate.

The foregoing experiments bring additional evidence to support the conception that the adversive syndrome induced by the intracarotid injection of the anticholinesterase drug di-isopropyl fluorophosphate is not caused by hyperactivity of the inner ear, but that, rather, its mechanism is central in origin. This opinion is further substantiated by work done in this laboratory,[14] in which it was shown that injections of di-isopropyl fluorophosphate into a common carotid artery bring about changes in the electrocorticogram and the cholinesterase activity of various cerebral structures, particularly in the caudate nucleus of the side into which the injections had been made with the smaller doses that produced turning.[7b] When excessive doses of di-isopropyl fluorophosphate are injected, the adversive syndrome progresses to a seizure, which can again be demonstrated by the electrocorticogram and by further decreases in cerebral cholinesterase activity. If it can be agreed on that the origin of action of di-isopropyl fluorophosphate in the production of the adversive syndrome is central then dinenhydrinate may exert its ameliorating

13. Mettler, F. A.: Relation Between Pyramidal and Extrapyramidal Function, A. Research Nerv. & Ment. Dis., Proc. **21**:150-227, 1942.

14. Hampson, J. L.; Essig, C. F.; Willis, A., and Himwich, H. E.: Effects of Di-Isopropyl Fluorophosphate (DFP) on Electroencephalogram and Cholinesterase Activity, Electroencephalog. & Clin. Neurophysiol., 1950, to be published. Freedman and Himwich.[8] Gay and Carliner.[9]

influences on central rather than peripheral structures. Additional confirmation for the central action of dinenhydrinate can be afforded by its power to neutralize the grand mal–like electrical patterns. During the studies of the effects of di-isopropyl fluorophosphate on the brain wave, it was observed that dinenhydrinate corrected the abnormal brain waves.[15] These studies of the influence of dinenhydrinate, together with the evidence presented here, indicate localization of the effect of dinenhydrinate to central structures and, in the case of the adversive syndrome, to subcortical cerebral areas associated with vestibular functions. A comparison between the effects of morphine and dinenhydrinate may illustrate their modes of action. Morphine probably does not affect the pain receptors but alters the interpretation of pain. Likewise, dinenhydrinate may not affect the peripheral receptors but, rather, may alter the central responses to vestibular stimuli.

SUMMARY AND CONCLUSIONS

A syndrome including compulsive circling, caused by intracarotid injections of di-isopropyl fluorophosphate, was subjected to study in order to analyze the underlying mechanism. Both peripheral and central elements were evaluated. Though the syndrome may be evoked by changes in vestibular receptors, as illustrated by the effects of their cocainization, when di-isopropyl fluorophosphate was used locally in the ear, the characteristic behavioral changes could not be aroused in either rabbits or cats. The application of this drug to the round window or direct injection into perilymph failed to evoke forced circling. These experiments, therefore, indicate that the vestibular receptors are not primarily involved in the syndrome as induced by di-isopropyl fluorophosphate. They also suggest that the vestibular receptors are not cholinergic in nature.

In contrast, the central portion of the mechanism is stimulated by di-isopropyl flurophosphate, which presumably allows the existence of an excess of acetylcholine in the brain, and the resulting abnormal behavior is corrected by atropine. Dinenhydrinate, which exhibits atropine-like properties, is effective against the excessive acetylcholine. It is suggested that this atropine-like action, exerted on subcortical structures, is the basis for the therapeutic influence of dinenhydrinate on this syndrome. In any event, the experiments demonstrate that dinenhydrinate and diphenhydramine hydrochloride prevent compulsive turning in rabbits when administered before di-isopropyl fluorophosphate and correct the abnormal behavior when given after it.

15. Esmond, W. C.; Johns, R. J.; Bales, P. D.; McCauley, A., and Himwich, H. E.: Convulsant and Anticonvulsant Effects of Some Antihistamines, Electroencephalog. & Clin. Neurophysiol., 1950, to be published.

HEATING OF THE HUMAN MAXILLARY SINUS BY MICROWAVES

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THE ABILITY to heat the human paranasal sinuses has been a long sought after objective. Limited success has been obtained with the use of infra-red, conventional and short wave diathermy as the heating agent.[1] Since the end of World War II microwaves have been added to the heating armamentarium. Perfection of the magnetron tube made the generation of microwaves possible.

In 1948, Osborne and Frederick [2] reported the use of microwaves to produce a marked temperature rise in the frontal sinus of the dog. In view of these experiments, it was thought advisable to undertake experiments to determine whether the intra-antral temperature of human subjects might be similarly increased.

Since 1947 experimental data have been presented to show that microwaves can produce a significant rise in temperature in living tissues. Microwaves differ from short wave diathermy currents in that, apart from their extremely high frequency, they possess optical properties so that they can be reflected, refracted, diffracted and focused. Because the microtherm, a microwave generator, seemed to offer a new approach to the problem, this experimental study was undertaken.

In 1936, Andreen and Osborne [1] attempted to increase the intra-antral temperatures of 17 patients who suffered from acute and chronic sinusitis but who had not undergone antrotomy. The heating effect of seven different agents was studied. After anesthetization with 10 per cent cocaine, entry was made to the antrum by introducing a trochar through the inferior meatus. Temperature readings were taken with a thermocouple until they were stabilized, then some form of heating

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1. Andreen, M. A., and Osborne, S. L.: Measurements of the Temperature of the Maxillary Sinus After Treatment by Various Methods of Heating: A Comparative Study, Arch. Otolaryng. **26**:331-337 (Sept.) 1936.

2. Osborne, S. L., and Frederick, J. N.: Microwave Radiations: Heating of Human and Animal Tissues by Means of High Frequency Current with Wavelength of Twelve Centimeters (The Microtherm), J. A. M. A. **137**:1036-1040 (July 17) 1948.

was applied for twenty minutes, after which temperature measurements were recorded again. Highest temperatures were secured with high frequency currents. With conventional diathermy the average initial temperature rose from 98.2 to 99.1 F., a rise of 0.9 F. The electromagnetic field produced a rise from 98.5 to 99.1 F., a gain of 0.6 F.

Rosenwasser and Bierman [3] in 1937 attempted to increase the intra-antral temperatures by means of short wave diathermy in 2 patients who had patent antrotomy openings. They used three machines generating currents of 6, 15 and 16 meters respectively. Long, flexible thermocouples inserted through the antrum window were used to record the temperatures. The average antral temperature of the two subjects before heating in ten trials was 93.99 F. In four of these ten experiments no anesthesia was used, and after heating a final average temperature of 94.4 F. was secured, or a gain of 0.41 F. In the remaining six experiments an anesthetic agent of 5 per cent cocaine-epinephrine solution was used, and the gain after heating was more marked, 0.81 F. The current was given to the point of "comfortable heat tolerance" and allowed to flow twenty to thirty minutes.

In further experiments, Rosenwasser and Bierman [3] made temperature studies before and after diathermy treatment on 44 patients with operative openings in the anterior wall of the sphenoid sinus. No anesthesia was used, and diathermy was given "to the point of comfortable tolerance" for fifteen minutes. After cessation of the treatment, the electrodes were removed and the thermocouple inserted through the sphenoidal opening. They found the average temperature before heating to be 96.79 F. and after the treatment 98.1 F., a gain of 1.31 F.

An experiment similar to the one described was done by Hollender [4] in 1939. He selected 20 cases of acute exacerbation of maxillary sinusitis in which the taking of temperatures through an antrum window was feasible. No anesthetic agents were used, and short wave diathermy was given over a fifteen minute period with air-spaced electrodes. In 10 instances the intra-antral temperature was elevated 0.6 F. to 1.5 F., in 4 no change occurred and in 6 the temperature fell 0.4 to 1 F.

The only experiments reported to date on the use of microwaves for heating of the paranasal sinus are those of Osborne and Frederick.[2] These workers, using microwaves of approximately 12.5 cm., irradiated the frontal sinus of 6 dogs. The animals were under pentothal sodium anesthesia, and a thermocouple was inserted through the medial end

3. Rosenwasser, H., and Bierman, W.: Effect of the Short Wave Current on the Temperature of the Paranasal Sinuses, Arch. Otolaryng. **25**:555-559 (May) 1937.

4. Hollender, A. R.: Short Wave Diathermy in Treatment of Nasal Sinusitis, Arch. Otolaryng. **30**:749-754 (Nov.) 1939.

of the supraorbital border of the frontal bone into the cavity of the sinus. The energy was reflected from a rectangular director placed at a 2 inch (5.08 cm.) skin director distance. Their results were tabulated as follows:

	Average Temperature Before	Average Temperature After	Maximum
Frontal sinus	96.1	106.1	106.9
Rectal	101.2	101.9	.....

This is the highest temperature increase, 10 F., so far reported in the literature.

EXPERIMENTAL PROCEDURE

All temperatures were measured with a thermocouple made from constantan and copper wires soldered into the tip of a Holmes antrum catheter. The thermocouples were attached to a potentiometer of the requisite sensitivity.

The Microtherm was used as a generator of microwaves. This instrument operates at a frequency of 2,400 to 2,500 megacycles on a wavelength of approximately 12.2 cm. with a maximum power output of 125 watts. The entire radio-frequency circuit consists of an air-cooled magnetron oscillating tube with a coaxial cable connected to a director. The shape and size of the director determine the zone of tissues to be treated.

A total of thirteen experiments on 9 patients were performed. Three of the patients were female and 6 male, and all suffered clinically from low grade sinusitis of a chronic (months to years) nature. None of the patients had undergone antrotomy for the treatment of their disease.

The experiments were carried out without anesthesia as follows: The thermocouple was inserted into the anatomic ostium of the maxillary sinus. Temperature recordings were commenced at once, continued until a base level was reached and maintained for a period of twenty minutes. The thermocouple then was removed and the maxillary sinus irradiated with microwaves for a period of twenty minutes. In the majority of experiments, a circular 4 inch (10.16 cm.) director was used. Because Richardson, Duane and Hines [5] and Daily, Wakim, Herrick and Parkhill [6] have reported the development of lenticular opacities in the eyes of rabbits and dogs following irradiation by the microtherm, it was deemed advisable to shield the eyes on the irradiated side, a flat metal cone being used to reflect the microwaves from the eye

5. Richardson, A. W.; Duane, T. D., and Hines, H. M.: Experimental Lenticular Opacities Produced by Microwave Irradiations, Arch. Phys. Med. **29**:765-769, 1949.

6. Daily, L., Jr.; Wakim, K. G.; Herrick, J. F., and Parkhill, E. M.: Effects of Microwave Diathermy on the Eye, Am. J. Physiol. **155**:432, 1948.

region (fig. 1). The director was placed at a distance of 2 inches (5 cm.) from the skin overlying the sinus and the generator operated at 80 to 85 per cent of its total output, at which setting the heat tolerance of the patient was reached. It is not possible, however, to determine the energy absorption (dosage) for any individual patient.

Fig. 1.—Application of microwaves to the left maxillary sinus. The eye has been shielded with a metal cone.

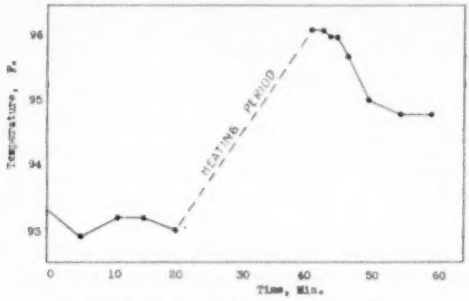

Fig. 2.—A typical graph of one experiment.

Tolerance was the only absolute guide. Therefore, we have no assurance that each person received the same dose of irradiation.

Immediately after the cessation of the irradiation, the thermocouple was reinserted into the maxillary ostium and temperatures recorded until a base level was reached. Approximately a two to three minute period elapsed between the cessation of the irradiation and the first temperature recording.

RESULTS

Of the thirteen experiments, five were not included in the tabulation of the results because either it was not certain that the thermocouple was inserted into the maxillary ostium (3 cases) or when the data were reviewed it appeared that insufficient time had elapsed for a pretreatment base level of the intrasinus temperature to be obtained (2 cases). Figure 2 shows a typical graph of the temperature changes and figure 3 the composite graph made from the average temperatures of the eight experiments five, ten, fifteen and twenty minutes after the cessation of

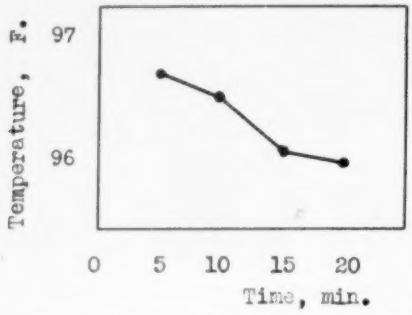

Fig. 3.—Composite graph of the eight experiments showing the temperature five, ten, fifteen and twenty minutes after the cessation of the heating period.

Statistical Data

Mean before heating	96.11 F.
Mean after heating	97.46 F.
Difference between means	1.35 F.
S. D. difference between means	1.48
S. E. difference between means	0.56
t ratio	2.41

the heating period. The accompanying table gives the statistical data on all experiments. The statistical method of paired comparisons was used.

While this temperature rise of 1.35 F. is not pronounced, it is statistically significant on a 5 per cent level. Thus, a change of this magnitude might occur by chance once in twenty such experiments.

COMMENT

The average temperature rise of 1.35 F. is higher than that obtained by Andreen and Osborne,[1] who reported a gain of 0.9 F. in intrasinus temperature after using short wave diathermy. The work of Rosen-

wasser and Bierman [3] is not comparable to the present work because the sinuses experimented on had operative, permanently patent communications between the nasal lumen and the sinus.

Osborne and Frederick,[2] however, after irradiating dogs' frontal sinuses with microwaves in an experiment similar to the present one, have observed an average gain in intrasinus temperature of 10 F. in 6 dogs. In the present work on human beings, an average gain of only 1.35 F. was obtained. This difference is probably partly explained by the following factors:

1. Anatomic factors. The average volume of 23 dogs' frontal sinuses measured by one of us (J. J. B.[7]) was 2.8 cc., whereas that of the adult human maxillary sinus, according to Schaeffer, is approximately 14.75 cc.[8] This figure for the human maxillary sinus was obtained from cadavers and thus represented the contents of the bony cavity without mucous membrane. On the other hand, measurements of the dogs' frontal sinuses were made on anesthetized dogs or dogs dead but a few minutes, in which case the mucous membrane was not disturbed. However, though the discrepancy in size of the two sinuses may not be as great as indicated, the volume of the human sinus treated was approximately four and a half times larger than that of the dog's sinus and would require more energy to raise the temperature by a unit.

2. Thickness of bone. The bones of the anterior wall of 23 dogs' frontal sinuses measured [7] were found to be approximately 2 mm. thick. Schaeffer [8] stated that the wall of the human adult maxillary frontal sinus usually varies from 5 to 8 mm. in thickness. Thus, the anterior wall of the human maxillary sinuses experimented on offer a greater obstruction to the passage of microwaves than does the anterior wall of the dog's frontal sinus reported on by Osborne and Frederick.

3. Position of thermocouple. The human ostium maxillaris is located in the nasoantral wall near the superior border and at or slightly ventral to the midpoint in a dorsal-ventral direction. In the present work, the thermocouple was located in the ostium maxillaris. Osborne and Frederick inserted the thermocouple just below the anteroventral wall of the dog's frontal sinus, and thus it was located relatively nearer the source of microwaves than in the present experiment.

4. Blood supply. The final effect of the blood supply to the dog's frontal sinus and the human maxillary sinus is unknown.

5. Anesthesia. The differences in anesthesia (no anesthesia in the human being and general anesthesia in the dog) might be a contributing factor. It is known that the body temperature of dogs under general anesthesia rises.

7. Ballenger, J. J.: Unpublished data.

8. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Sons & Co., 1920.

6. Presence of disease. In the work reported by Osborne and Frederick the dogs were free from gross disease, whereas the patients being reported on suffered from low grade chronic sinusitis. The latter might have the effect of thickening the sinus lining mucosa, increasing the sinus secretions and sclerosing the bony sinus walls.

SUMMARY AND CONCLUSIONS

An attempt has been made to elevate the temperature of the intramaxillary sinus in 8 patients suffering from low grade chronic sinusitis by use of microwaves. An average elevation of 1.35 F. was obtained. On the surface, a gain as small as this does not appear of importance therapeutically, but other factors, not measured in the present experiment, must be considered, such as the increase in blood flow. It is conceivable that marked physiologic effects such as increased active hyperemia could occur without a notable increase in temperature affecting the metabolism of the area treated and thus produce desirable therapeutic results. This experimental work is being augmented by a clinical study of the effect of microwaves.

SPEECH REHABILITATION OF PATIENTS WITH CLEFT PALATE

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IN THE speech clinic of the Manhattan Ear, Nose and Throat Hospital, my associates and I have had the opportunity of observing and training the speech of 43 patients afflicted with the congenital deformity of cleft palate. In all but 6 patients the cleft included the lip, and in 1 patient it even extended up to the eye. In 33 of these patients the palate had been closed by operation; 6 were seen before any surgical intervention on the palate, and 4 were wearing obturators. Their ages ranged from 3½ to 47 years. Speech tests were given to children as young as 18 months, but effective speech training cannot start before the child reaches about nursery school age. Most of the patients came to the clinic for rehabilitation training twice a week.

Congenital shortening in association with cleft palate is a deficiency which causes one of the most serious impediments to the development of normal speech. Congenital shortening is frequently found with a fissure of the alveolar process, but is not always associated with it, since one cleft may be present without the other. The cleft in the lip may be limited to a small groove or may extend up to the nose and eyelids.[1] Following is a list of the abnormal anatomic types of cleft:

(1) Prealveolar cleft, (*a*) unilateral and (*b*) bilateral.

(2) Alveolar cleft, (*a*) unilateral and (*b*) bilateral. (A bilateral alveolar cleft produces a protrusion of the premaxillary bone and is usually associated with double cleft palate.)

(3) Postalveolar cleft, (*a*) hard and soft palate, in part or total; (*b*) soft palate, in part or total, and (*c*) submucous fissure of the hard palate, in part or total.

The palate cleft may be classed as unilateral if it extends backward and if the nasal septum is attached to one side of the cleft only; it may be classed as bilateral if the nasal septum is not attached to either side but is a separate structure in the middle with a cleft on each side.

In addition to these conditions, the cleft may extend over the prealveolar, alveolar and postalveolar region or may be limited to the first and second region or to the second and third.

From the Speech Clinic of the Manhattan Eye, Ear and Throat Hospital, service of Dr. Daniel S. Cunning, Surgeon Director.

1. Learly, D. L., in Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat, and Ear, Including Bronchoscopy and Esophagoscopy, Philadelphia, W. B. Saunders Company, 1945.

Feeding difficulties make it advisable to repair the cleft of the lip and the alveolus a few weeks after birth, whereas the repair of the palatal

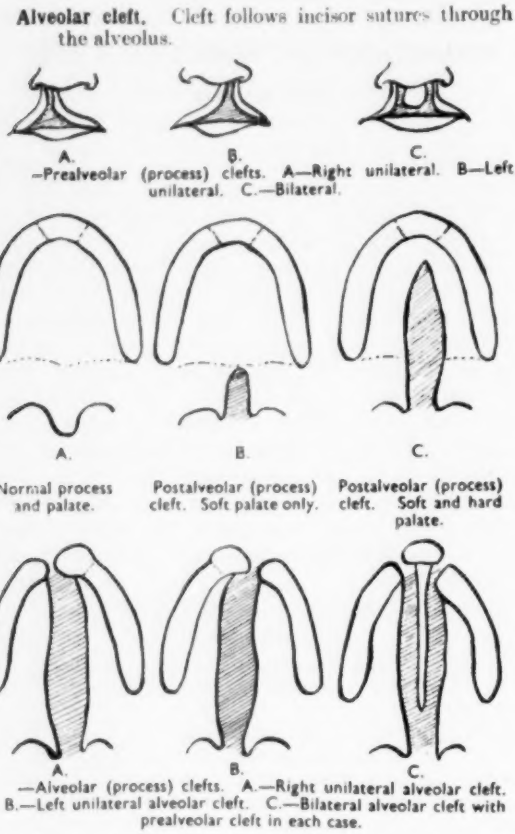
Alveolar cleft. Cleft follows incisor sutures through the alveolus.
A.
B.
C.
—Prealveolar (process) clefts. A.—Right unilateral. B—Left unilateral. C.—Bilateral.
A.
B.
C.
Normal process and palate.
Postalveolar (process) cleft. Soft palate only.
Postalveolar (process) cleft. Soft and hard palate.
A.
B.
C.
—Alveolar (process) clefts. A.—Right unilateral alveolar cleft. B.—Left unilateral alveolar cleft. C.—Bilateral alveolar cleft with prealveolar cleft in each case.

Fig. 1.—Diagrams of cleft palate. From Morley,[3] with permission of E. & S. Livingstone, Edinburgh.

malformation should be delayed until the child is in his second year[2]; some authors even recommend waiting until the child is 5 years or older.[3]

2. Conway, H.: One-Stage Push-Back Operation for Congenital Insufficiency of Palate, Oral Surg., Oral Med. & Oral Path. **1**:640, 1948.

3. Dorrance, G. M., and Shirazy, E.: The Operative Story of Cleft Palate. Philadelphia, W. B. Saunders Company, 1933. Parker, D. B.: Surgical Treatment of Clefts of Palate and Lip, Am. J. Surg. **4**:385, 1928.

To explain the physiology of articulation one may compare the oral cavity to a kettle in which valves regulate the air pressure, for air pressure is a *sine qua non* for normal enunciation of most of the human speech sounds. Four groups of muscles functioning in different areas of the mouth control the action of these valves.

The orbicularis oris muscle is used in pronouncing *w*, *b* and *p*; and the upper incisors are placed on the lower lip to formulate the sounds of *f* and *v*.

The tip of the tongue articulates at the alveolar process and makes possible the enunciation of *t*, *d*, *sh*, *s*, *th*, *l* and the rolled *r*.

The back of the tongue closes the oral cavity at the middle portion of the palate for the enunciation of hard *g* and *k*.

The superior constrictor pharyngis and the levator palati muscle form the palatopharyngeal septum which when in action completely separates the oral cavity from the nasopharynx and thus prevents the escape of air into the nose. This septum is essential for the denasalization of the vowels and is the basis of speech without hyperrhinolalia.

To understand these mechanisms, it is essential to remember that the walls of the pharynx consist of the superior, middle and inferior constrictor muscles of the pharynx; it is the first—the superior constrictor pharyngis—which, on contraction, forms a horizontal pad known as Passavant's bar. The constrictor muscles of the pharynx also serve deglutition and are mainly innervated by the vagus nerve. The levator palati and tensor palati muscles raise the soft palate and open the orifices of the eustachian tube. The palatoglossus muscle in the anterior pillar and the palatopharyngeus muscles in the posterior pillar draw the palate downward. The motor innervation for these muscles is supplied by the vagus and the cranial portion of the accessory nerve which carry motor fibers to the soft palate. Efferent fibers of the facial nerve enter the greater superficial petrosal nerve and reach the sphenopalatine ganglion, whose fibers go to the mucous membrane of the roof of the mouth,[4] whereas the tensor palati are innervated by the internal pterygoid nerve (the tensor veli palatini), which consists of efferent motor fibers of the mandibular branch of the trigeminal nerve.

The sensory nerve of the palate, the sphenopalatine, is a branch of the maxillary division of the trigeminal nerve; it is the glossopharyngeus which carries the afferent sensations of the pharynx.

4. Strong, O. S., and Elwyn, A.: Human Neuroanatomy, Baltimore, Williams & Wilkins Company, 1943.

It is generally true that whenever a congenital or traumatic malformation interferes with the physiologic function of one of the previously mentioned groups of muscles, the speech sounds for which they are responsible will be defective or will be replaced by some other sound.

In normal circumstances a child begins to express himself in articulate language when he is approximately 15 months old.[4a] At this age he has developed the faculty of coordinating the various muscles essential for articulation and has acquired, through acoustic and visual stimuli, the speech pattern of his environment. At the age of 4 he will have built up a vocabulary of about two thousand words and mastered the ability to arrange these words into correct grammatical sentences.[4] After the age of 4, speech habits are firmly established, and only specialized training can change them.

The child with cleft palate, too, attempts to imitate the speech patterns of his environment, but as a consequence of his deformity he is obliged to compensate for the speech sounds he cannot make by substituting others. He becomes accustomed to identifying the sounds he hears himself uttering with the sounds he endeavors to copy. Contrary to the stutterer, who suffers greatly because of his speech impediment, the child afflicted with a congenital cleft palate is completely unaware of any difference between his speech and the speech patterns he hears around him.

The speech examination for children of preschool age consists in showing them simple pictures, which are chosen with regard to the three articulation zones, the labial, the dental and the palatal, and to observe the sounds these zones produce. Thus, to test the labial sounds, a picture of a bell, pig, mouse, wheel or fork is shown; to test the dental, pictures of a dog, cat, table, scissors, spoon, pussycat, shoe or chair is shown. The palatal sounds are tested by a picture of a goose, cow or car.

It goes without saying, that, in order to get exhaustive speech evaluation, all speech sounds have to be tested isolated and in all potential sound combinations. Such thoroughness is unnecessary in cases of cleft palate except for purely diagnostic purposes, since all defects will obviously manifest themselves during rehabilitation exercises.

The striking characteristics of cleft palate speech are hyperrhinolalia (nasalization) and dyslalias (sound substitutions and sound omissions). The stopped or plosive consonants *d*, *t*, *g* (as in garden) and *k* are replaced by a glottal stop, and *s* and *sh* are blown through the nasal cavity; they sound like a snort and are accompanied with a nasal grimace (fig. 2). The glottal stop, the multiform dyslalias and the hyperrhinolalia transform the speech of the patient into an unintelligible jumble of

4a. Buehler, K.: The Mental Development of the Child: A Summary of Modern Psychological Theory, London, Kegan, Paul, Trench, Trubner & Co., Ltd., 1930.

sounds. Even when the prealveolar cleft has been sutured, the orbicularis oris has to learn to adjust to its task as articulation muscle, since the *p*, *b*, *m* and *w* are not enunciated.

The basis of speech training of these patients is instruction in preventing the escape of air into the nose; the way to accumulate air pressure in the oral cavity, and the use of such pressure in articulation. The duration and results of therapy will depend on several factors: the length and mobility of the sutured palate; the compensations and sound substitutions which have become a habit, and the intelligence of the patient and his hearing acuity.

Fig. 2.—Boy with congenital deformity of sharp palate, in association with cleft, enunciating the nasal *s* with the typical nasal grimace.

The restoration of normal speech is the criterion of successful treatment for cleft palate; nevertheless, the factors just mentioned may jeopardize this result seriously. After a few months of training, whenever the patient fails to improve his consonants, shows no change in the nasalization of the vowels and continues to produce his *s* and *z* with overwhelming nasal escape, additional surgical procedure is indicated. This second operation, originally introduced by Passavant in 1863 and improved in 1920 by Ganzer in Germany and Sir Harold Gillies in England, was described in the United States in 1925 by Dorrance,[5] who called it the pushback operation because, after dissection,

the loose tissues of the entire palate are displaced backward, producing lengthening of the soft palate. Other surgeons who have made important contributions to the technic of this procedure are Victor Veau, of Paris, who published his book "Division Palatine" in 1931 and called the operation "Veau Operation." In 1937 Wardill synthesized the advantages of the Gillies, Veau and Dorrance technics and combined this operation with pharyngoplasty to obtain the velopharyngeal occlusion, the physiologic mechanisms for speech without hyperrhinolalia.

To insure the best anatomic and physiologic condition of the sutured palate, Morley,[5] after a careful evaluation of all the recent methods of palatal surgery, worked out five points, which must be included in modern cleft palate repair. Following are these five points.[5]

1. The nasopharynx is narrowed or the posterior wall of the pharynx is brought forward to assist in closure of the sphincter.

2. The two halves of the palate are sutured in such a way that the muscles are united, as in the normal palate, and the length of the soft palate is increased as far as possible.

3. The suturing of the soft palate must be done without tension, and for this purpose structures such as the hamular processes and the posterior palatine artery may be destroyed, care being taken that the blood supply to the palate is maintained or the tissues will not survive.

4. Suturing is carried out so as to avoid as far as possible the formation of scar tissue on both the nasal and oral surfaces. This would otherwise cause thickening and lack of flexibility in the soft palate.

5. If there is a scarcity of tissues the hard palate is sacrificed for the benefit of the soft palate, the hard palate defect being closed later by a dental plate.

In the speech clinic of the Manhattan Ear, Nose and Throat Hospital, 90 per cent of the patients are children, and for them the training has to be disguised as play, for their cooperation is essential for success. Games devised to teach them to prevent the escape of air into the nose and to expel it through the mouth instead are blowing games, such as blowing bubbles in a glass of water. To prevent the nasal escape of air during these games the child's nose is held between thumb and forefinger. Subsequently, the fingers are removed and the child is trained to achieve a similar result without them.

Later, the patient is taught to blow a small piece of paper which has been stuck to his lips by an explosive release of air accumulated in the mouth. This is an easy method for mastering the sounds *b* and *p*. As soon as these sounds are correctly enunciated, they are practiced in

5. Morley, M. E.: Cleft Palate and Speech, Baltimore, Williams & Wilkins Company, 1945.

syllables, such as *pa, pe, pi, po, poo, ba, be, bi* and *boo*. Usually, after the second or third session, it is advisable to drill the child in simple words which begin alternately with these letters. Schoolfield's book[6] provides an adequate vocabulary for these drills. The repetition of simple stories and nursery rhymes is very helpful in building the newly acquired sounds into the spontaneous speech pattern.

The enunciation of *d* and *t* is achieved by expelling a piece of paper stuck between the tip of the tongue and the alveolar ridge. A tongue

Fig. 3.—Diagram showing use of the tongue depressor for the production of the sound *k* and hard *g*.

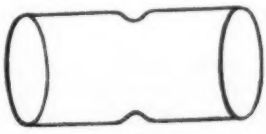

Fig. 4.—Drawing of glass tube to fix the tongue in position for the correct enunciation of *sh*.

depressor to keep the tongue with its tip on the floor of the mouth is used to produce *g* and *k*. One of the patient's hands is placed between the therapist's throat and chin, the other on his own throat; then he is asked to pronounce *t* while the teacher enunciates *k*. The glottal stop—a sound produced by explosion through the vocal cords—is the sound substitution for hard *g* and *k*. The glottal stop forms a deep-rooted habit and proves one of the most stubborn as well as one of the

6. Schoolfield, L. D.: Better Speech and Better Reading: A Practice Book. Boston, Expression Co., 1937.

most difficult to eradicate. The procedure of reeducation is nevertheless identical to that of the other speech sounds; as soon as a correct hard *g* (as in go) and *k* has been achieved in isolated enunciation, they are practiced in such syllables as *ga, ge, gi, go* and *goo; gla, gle, gli, glog* and *gloo; gra, gre, gri, gro* and *groo;* and the *k* in *ka, ke, ki, ko* and *koo; ska, ske, ski, sko* and *skoo.* Later, they are used in words and sentences. It may require three months or longer of persistent drilling before the patient is able to apply these two sounds automatically in spontaneous speech.

Placing the glass tube of an eyedropper at the lower incisors of the patient is a good way to teach him to blow the sounds of *s* and *z*. He must succeed in directing the air current through this small glass tube.

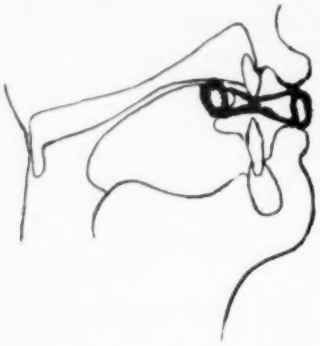

Fig. 5.—Diagram showing position of glass tube in the mouth.

The physiology of *s* and *z* requires sustained air pressure forced through a very small opening, an impossible feat in the presence of an open outlet between the pharyngeal wall and the soft palate.

A glass tube, ½ inch (1.27 cm.) in diameter, fixes the tongue in the central position and enables the patient to pronounce a concentrated *sh* in a similar manner.

A correct sounding, sharp *s* is a definite indication of a palate long and mobile enough to act as a perfect septum between nasal and oral cavities, which prevents the passage of air from pharynx into the nose; the formation of *g* and *k*, however, does not require complete velo pharyngeal occlusion.

Three case histories are presented which will best illustrate the therapeutic methods used in the training of speech.

REPORT OF CASES

CASE 1.—The unilateral cleft palate in a patient aged 15 was repaired when he was 2 years old. Before he was referred to the speech clinic, he had undergone a Dorrance pushback operation. The distance between the pharyngeal wall and posterior end of the palate was a little less than 1 cm. Insufficient mobility of the palate could be observed during the enunciation of vowels. Further speech analysis disclosed a marked hyperrhinolalia (rhinolalia aperta) and a weak articulation of the labial and dental plosives. The palatal sounds were represented by a glottal stop, and *s* was replaced by a snore.

To train the patient in the use of his soft palate, he was instructed to push his fists hard, which were folded at the level of the pectus, downward from there, synchronizing this motion with the enunciation of the vowels *a*, *e*, *i*, *o* and *u*, and such syllables as *pa*, *pe*, *pi*, *po* and *pu*. These exercises are based on the physiologic law that when one set of muscles are pulled in one direction, another set automatically compensates by moving in the opposite direction.[7] These pushing exercises promoted the use of the palatopharyngeal septum. The treatment continued by closing the patient's nose with thumb and forefinger and instructing him to inhale with his mouth wide open. The therapist demonstrated on her velum what she wanted him to do and gave him a mirror so that he could observe his own palate. All these exercises were means to increase the mobility of the velum, not only during inhalation but also during exhalation. The hyperrhinolalia soon disappeared completely, and by applying methods already described, our patient subsequently acquired good labial, dental and palatal plosive sounds and made correct use of them in his spontaneous speech.

In learning to enunciate *s* and *z*, the patient was given a stethoscope[8] and listened to the therapist speaking into the ball of the stethoscope words ending in *s* or *z*. This procedure had the purpose of adding to the patient's visual and kinesthetic impressions a very precise auditory effect. After three months of biweekly treatment, these sounds, too, were correctly fitted into his spontaneous speech.

About this time we were told by the patient's mother that his high school teacher had commented favorably on the improvement in his speech. The patient himself revealed that his classmates, who had formerly ridiculed and imitated him, had stopped doing so and seemed no longer aware of any abnormality in his speech. After the fourth month the treatment was discontinued because of the entirely satisfactory results.

CASE 2.—A boy aged 11 was referred from his school for speech rehabilitation. His congenital deformity consisted of prealveolar, alveolar and postalveolar unilateral cleft, associated with shortening of the palate. His cleft lip had been repaired during his first weeks of life and the palate sutured when he was 2 years. Since then, the mother reported, the boy had undergone two more operations in an attempt to improve his speech, the last one before he entered school. The family lived in New Jersey and the patient came to New York especially for speech training.

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7. Froeschels, E.: Lehrbuch der Sprachheilkunde (Logopädie) für Aerzte, Pädagogen und Studierende, Leipzig, F. Deuticke, 1931.

8. Freud, E. D.: Un nouvel appareil pour exercise auditifs, Pract. oto-rhino-laryng. **4**:169, 1942.

The examination of the oral cavity disclosed a soft palate and velum of sufficient mobility; the distance between pharyngeal wall and soft palate was 0.5 cm. There was a postalveolar hole 2 cm. in diameter in the hard palate. The upper incisors were missing. The speech test disclosed clear denasalization of vowels; *s* and *z* were enunciated interdentally, the tongue protruding through the opening left by the missing incisors, and sounded identical with *th*. They were distorted by the noise of air which escaped through the nose and were accompanied by a nasal grimace. See figure 2.

D and *t* were represented by glottal stops, whereas *k* and *g* were correctly formed. Although the child was cooperative and intelligent, the dental sounds defied all methods of correction until the postalveolar cleft was closed with a piece of adhesive tape. As long as the adhesive tape stuck to the palate, the dental plosives and the *th* could be easily formed. A dental plate was recommended to cover the opening and to replace the two missing incisors.

As soon as the boy returned to the speech clinic wearing his obturator, which covered the postalveolar opening completely, he was able to learn the enunciation of *d* and *t;* even *s* and *th* had lost the distorting noise made by the nasal escape of air. After a few weeks of additional training, the speech improved remarkably and the patient no longer required speech therapy.

Case 3.—This patient was a woman aged 43. She had never had any surgical intervention, but she wore a solid obturator that completely closed the unilateral cleft of the hard and soft palate and maintained contact with the remaining palatovelar muscles during phonation and deglutition. At its posterior aspect the obturator was equipped with a hinge that ended at approximately 2 cm. from the pharyngeal wall. In spite of her condition, the speech of this patient was nearly devoid of hyperrhinolalia, a fact which could be explained only by close observation of her pharyngeal wall during enunciation of the vowel *a*. This patient had developed the constrictor pharyngis muscle in such a way that the pharyngeal wall as a whole protruded forward and narrowed the gap between the end of the prosthesis to a distance less than 1 cm. It seemed as if the pharyngeal wall had attempted to compensate for the congenital deficiency of the palate. Her speech defect consisted mainly of the glottal stop substituted for hard *g* and *k*.

The patient refused any suggestions for surgical intervention. She stated that she just wanted speech training, explaining that people had difficulty in understanding her, probably because of her foreign accent. She was incredulous when it was pointed out to her that the production of the palatal sounds by means of a glottal stop was the cause of her distorted speech, and she brought her family doctor to her lesson so that he might check whether the new sounds she produced were "the real thing." Again and again she asked, "Can I really talk this way? It sounds so affected." She was finally convinced when her husband and her friends made spontaneous remarks about how much her speech had improved. It took four months of biweekly training before sounds she had learned were automatically introduced in her spontaneous speech.

The observations made on patients such as these lead to the obvious conclusion that the physiologic premise for rehabilitation of good speech is the potential occlusion between palate and pharyngeal wall during enunciation. Nevertheless, whenever, in cases of congenital malforma-

tion, surgical procedure was able to build a new palate long and mobile enough to provide for this function, good speech could be acquired only if the operation was performed at an age before definite speech patterns had been established. At a later age, surgical intervention must be attended by speech rehabilitation as the only means of erasing faulty speech habits and organizing speech patterns in harmony with physiologic laws.

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CANCER OF THE LARYNX CLASSIFIED IN THREE DIMENSIONS

An Aid in Management

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IN THE choice of a therapeutic modality the only consideration, of course, is the effect it will have on the patient. In spite of this recognized fact, whenever cancer of the larynx comes up for discussion the surgeon and the radiologist often take opposite stands.

I believe that this state is not the result of personal consideration. It stems from two factors. The first factor is that the surgeons are not thoroughly acquainted with the radiologists' statistics and, perhaps, vice versa, and the second is that analysis of statistics are interpreted on the basis of an inadequate classification.

Cancer of the larynx is generally classified as intrinsic and extrinsic.[1] This classification is surgical rather than histologic. The intrinsic variety is limited to the true vocal cords and the ventricular bands. A cancer is considered intrinsic when it does not overflow the rim of the larynx. The extrinsic variety involves the aryepiglottic folds, epiglottis, arytenoid cartilages, retrocricoid and piriform sinuses.

It is obvious that this classification makes no allowance for depth penetration, which is clinically recognized by fixation of the vocal cords. Yet, depth penetration is very important in determining the choice of a method of treatment and prognosticating an end result. For example, Cody [1a], in reviewing 4,584 cases, stated that the rate of cure for a five year period was 76 per cent for treatment by laryngofissure and 67 per cent for irradiation. He stated later in the article that a fixed vocal cord was not considered a contraindication to irradiation. He did not state whether or not a fixed vocal cord was considered a contraindication to laryngofissure. However, the general consensus by laryngologists

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1. (*a*) Cody, C. C.: Treatment of Cancer of the Larynx, Laryngoscope **59**:621 (July) 1949. (*b*) Cutler, M.: Concentration Radiotherapy of Cancer of the Larynx: Study of 413 Cases, J. A. M. A. **124**:967 (April 1) 1944.

is that a fixed cord is a contraindication to laryngofissure. From this the conclusion may be drawn that the methods of treatment represent different clinical entities and should not be used for comparison.

Of 70 patients with tumors of the true vocal cord treated with irradiation by Cutler,[1b] he cited an over-all rate of cure for 60 per cent for a five year period. However, when his figures were broken down, it was noted that 30 of these patients had mobile vocal cords with an 80 per cent rate of cure; whereas those with fixed cords showed only a 20 per cent rate of cure for a five year period. Here, again, a false evaluation of the statistics can be made if such a classification is followed.

For total laryngectomies, Jackson [2] showed a 65 per cent rate of cure for a five year period by surgical removal and 51 per cent by irradiation. The extent of the depth of the lesion was not given, and therefore the statistics are of no value for comparison.

Because such statistics are confusing, I have used a classification which includes depth penetration as well as surface extension.

CLASSIFICATION OF CANCER OF THE LARYNX

Intrinsic Superficial.—The neoplasm so classified lies within the rim of the larynx and originates from epithelium with a basement membrane. It is considered clinically superficial because the musculature is not infiltrated, that is, the cords are not fixed.

Intrinsic Penetrating.—The neoplasm in this classification has invaded the muscular tissue, and the vocal cord is either partly or entirely fixed.

Extrinsic Superficial.—Such a neoplasm, like the intrinsic, originates in the larynx but has overgrown or spilled over the rim. The "mother" tumor originated in mucosa with basement membrane. It has not invaded the musculature, as evidenced by the mobility of the vocal cords.

Extrinsic Penetrating.—The neoplasm so classified is similar in every way to the extrinsic superficial type, but the cells have invaded the muscle and there is partial or total fixation of the vocal cords.

Extrinsic Hypopharyngeal.—The neoplasm in this classification originates in epithelium with no basement membrane. It is really a hypopharyngeal tumor. It is found on the epiglottis, aryepiglottic folds, arytenoid cartilages, postcricoid region and piriform sinuses.

With this classification in mind, the choice between surgical treatment and irradiation can be evaluated more intelligently. The following conclusions are those I have drawn from reviewing the work of Cutler, Jackson, Cody and my own experience.

2. Jackson, C. L.; Blady, J. V.; Norris, C. M., and Maloney, W. H.: Cancer of the Larynx: Five Year End Results in Series of Patients Treated Between 1930 and 1942, J.A.M.A. **138**:1080 (Dec. 11) 1948.

1. Intrinsic superficial lesions can be treated equally well by surgical removal or irradiation. However, such factors as availability of personnel must be considered. If surgical and radiologic personnel are equally available, irradiation has the advantage, for it preserves the organ.

2. Intrinsic penetrating lesions do not respond well to irradiation. With complete fixation of the vocal cord, Cutler [1b] got only a 20 per cent rate of cure for a five year period. His results are better with partial fixation, but I believe that they do not equal the results of surgical treatment. There is one point of caution I wish to make. A cord may appear fixed as a result of mechanical or inflammatory conditions. In cases in which the mobility of a cord is doubtful, trial irradiation can be instituted to reduce the bulk, and then the condition can be reevaluated.

3. Extrinsic superficial tumors are best treated by irradiation, with the exception of tumors that are the result of metastasis to the lymph nodes. Such tumors do not respond well to irradiation, but better results are achieved in dissections of the neck.

4. Extrinsic penetrating lesions should be treated surgically, with dissection of the neck if lymph nodes are present.

5. For extrinsic hypopharyngeal tumors poor results are achieved with any form of treatment.[3] Irradiation in such cases can be repeated, and the salvage is somewhat higher than that by operation.[4]

SUMMARY

A classification of cancer of the larynx is presented which considers depth as well as surface extension and origin of the growth in mucosa with and without basement membrane. A review of statistics from the literature is made in the light of this classification, and certain conclusions are drawn.

3. Baclesse, F.: Roentgenotherapy in Cancer of the Hypopharynx, J.A.M.A. **140**:525 (June 11) 1949.

4. Cutler,[1b] Jackson, Blady, Norris and Maloney.[2]

TRACHEOBRONCHIAL ASPIRATION FOLLOWING TONSILLECTOMY WITH GENERAL ANESTHESIA

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IT HAS been estimated that tonsillectomy is the most frequently performed surgical procedure in the United States.[1] The opinion apparently exists among both the laity and a large number of men in the medical profession that tonsillectomy is a relatively safe minor procedure. Actually, however, it has been our experience and also the experience of others[2] that the operation is not a minor surgical procedure, although it usually is not attended by any great rate of morbidity or mortality, if careful attention is given to detail. Certain details in the use of anesthesia, positioning, operative technic and immediate postoperative care become of prime importance when it is considered that pulmonary and other complications occur more frequently than we physicians should like to admit.

While the tonsillectomist is usually the first to deny the frequent occurrence of atelectasis, bronchopneumonia or pulmonary abscess following tonsillectomy, a study of the literature on these subjects should convince him otherwise. It has long been recognized that any operation with the use of a general anesthetic about the mouth and pharynx carries the potential danger of pulmonary complications. Tonsillectomy, being the most frequently performed operation in this region of the body, has therefore been held accountable by numerous authors in many instances for these serious sequelae.

The reports from England by Maxwell,[3] based on an analysis of more than 11,000 cases in which autopsy was performed, show single pulmonary abscess occurring in 199 instances, in only 2 of which the

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1. Tonsil Stumps, Current Comment, J. A. M. A. **134**:698-699 (June 21) 1947.

2. Boies, L. R.: Tonsillectomy in the United States, Ann. Otol., Rhin. & Laryng. **57**:352-363 (June) 1948.

3. Maxwell, J.: Lung Abscess, with Special Reference to Causation and Treatment, Quart. J. Med. **3**:467-522 (July) 1934.

abscess followed tonsillectomy; and Ranson and McGolrick [4] stated that abscess of the lung occurs in 1 out of every 2,500 cases with tonsillectomy.

On the other hand, reports by American authors differ greatly from those of Maxwell. Flick and associates [5] studied 172 patients of all ages with pulmonary abscess, with foreign body and neoplasm excluded as causes. In 121 instances (70 per cent) the abscess was postoperative. Of these, the abscess in 97 (56 per cent of the entire series) followed tonsillectomy; of this number, the abscess in 88 (51 per cent of all cases) occurred after the use of general anesthesia; whereas in only 4 (2.3 per cent) it occurred after the use of local anesthesia. In 5 cases the type of anesthesia induced was not reported. Hedblom [6] reported 692 cases of pulmonary abscess, 146 of which occurred postoperatively; the abscess in 47 cases (32 per cent) followed tonsillectomy, whereas in 14 (9.5 per cent) it occurred after dental extractions. The report does not include information on the type of anesthesia. Hedblom and Van Hazel [7] later reported a single case of post-tonsillectomy abscess occurring in the upper lobe of the right lung. They believed this condition was due to aspiration of infected blood during or after operation with general anesthesia.

Morrison [8] stated that in 20 to 30 per cent of all cases of pulmonary abscess the abscess followed tonsillectomy; whereas King and Lord [9] reported a figure of 38.6 per cent. In an analysis of the cases of abscess of the lung at the Massachusetts General Hospital during a five year period, Sweet [10] found that "of 125 cases 54, or 43.2 per cent, followed . . . tonsillectomy and 70, or 56 per cent, . . . had either tonsillectomy or some other operation in the mouth, nose, or throat. Only 14 . . . followed an operation on some other portion of the body."

4. Ranson, F. T., and McGolrick, L.: Lung Abscess Following Tonsillectomy, Brit. M. J. **2**:1020-1021 (Dec. 2) 1933.

5. Flick, J. B.; Clerf, L. H.; Funk, E. H., and Farrell, J. T.: Pulmonary Abscess: An Analysis of One Hundred and Seventy-Two Cases, Arch. Surg. **19**:1292-1312 (Dec.) 1929.

6. Hedblom, C. A.: Surgical Treatment of Acute Pulmonary Abscess and Chronic Pulmonary Suppuration with Especial Reference to Post-Tonsillectomy Type, J. A. M. A. **83**:1577-1581 (Nov. 15) 1924.

7. Hedblom, C. A., and Van Hazel, W.: Acute Post-Tonsillectomy Pulmonary Abscess of Right Upper Lobe, S. Clin. North America **12**:1145-1147 (Oct.) 1932.

8. Morrison, H.: Lung Abscess and Its Relation to Surgery of the Upper Respiratory Tract, New England J. Med. **218**:669-670 (April 21) 1938.

9. King, D. S., and Lord, F. T.: Certain Aspects of Pulmonary Abscess from an Analysis of Two Hundred and Ten Cases, Ann. Int. Med. **8**:468-474 (Oct.) 1934.

10. Sweet, R. H.: Lung Abscess: An Analysis of the Massachusetts General Hospital Cases from 1933 Through 1937, Surg., Gynec. & Obst. **70**:1011-1021 (June) 1940.

Brantigan and Looper [11] reported that in 10 out of 122 cases of pulmonary abscess the abscess followed tonsillectomy in their series, an incidence of 8.2 per cent.

At the Mayo Clinic, according to Norris and Landis,[12] out of 16,000 tonsillectomies performed with local anesthesia, pulmonary abscess followed in only 2 cases. No figures are given for the incidence with general anesthesia. However, the great difference in the reports from England and those of American authors may be explained on the basis of the more frequent performance of tonsillectomy with local anesthesia abroad. This explanation would seem to be borne out by the figures from the Mayo Clinic.

Haight and Ransom [13] stated that postoperative atelectasis is the probable precursor of pulmonary abscess. They emphasized the importance of the presence of bronchial secretions, together with a postoperative decrease in pulmonary ventilation and cough efficiency in the genesis of the condition.

Whereas Jackson and Jackson [14] stated that pulmonary abscess that occurs after a tonsillectomy develops as the result of a septic infarct from a blood-borne embolus, Myerson,[15] Iglauer,[16] Hara,[17] Ochsner and Nesbitt,[18] Eggston and Wolff [19] and May, Thoburn and Rosenberger [20] stated that aspiration was the source of pulmonary infection in such cases. In the study of this problem two questions arise which would lead one to favor the aspiration theory. First, infective emboli being multiple,

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11. Brantigan, O. C., and Looper, E. A.: The Etiology and Surgical Treatment of Lung Abscess: Importance of Lobectomy, South. M. J. **37**:199-207 (April) 1944.

12. Norris, G. W., and Landis, H. R. M.: Diseases of the Chest and the Principles of Physical Diagnosis, ed. 6, revised by S. S. Leopold, Philadelphia, W. B. Saunders Company, 1938, p. 609.

13. Haight, C., and Ransom, H. K.: Observations on the Prevention and Treatment of Postoperative Atelectasis and Bronchopneumonia, Ann. Surg. **114**:243-262 (Aug.) 1941.

14. Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat and Ear, Including Bronchoscopy and Esophagoscopy, Philadelphia, W. B. Saunders Company, 1945, p. 651.

15. Myerson, M. C.: Bronchoscopic Observations on the Cough Reflex in Tonsillectomy Under General Anesthesia, Laryngoscope **34**:63-68 (Jan.) 1924.

16. Iglauer, S.: Aspiration of Blood into the Larynx and Trachea During Tonsillectomy Under Local Anesthesia: A Contribution to the Etiology of Lung Abscess, Ann. Otol., Rhin. & Laryng. **37**:231-239 (March) 1928.

17. Hara, H. J.: Aspiration in Tonsillectomy: Comparative Merits of Posture and Other Factors, California & West. Med. **33**:628-638 (Sept.) 1930.

18. Ochsner, A., and Nesbitt, W.: Pulmonary Abscess Following Tonsillectomy: Preliminary Report, Arch. Otolaryng. **6**:330-337 (Oct.) 1927.

19. Eggston, A. A., and Wolff, D.: Histopathology of the Ear, Nose and Throat, Baltimore, Williams & Wilkins Company, 1946, p. 908.

20. May, R. V.; Thoburn, T. W., and Rosenberger, H. C.: Aspiration During Tonsillectomy: A Roentgenographic Study, J. A. M. A. **93**:589-592 (Aug. 24) 1929.

how is it possible that the resulting abscess is single? Second, if the embolic theory is correct, how can one explain the high incidence of post-tonsillectomy abscess of the lung following general anesthesia, as compared to the low incidence of this disease following local anesthesia? Accordingly, it would seem more logical to assume that aspiration is the source of infection in pulmonary abscess following tonsillectomy. Because of these considerations, a study was undertaken of a consecutive series of our own patients of all ages on whom a tonsillectomy had been performed with general anesthesia.

MATERIALS AND METHODS

Anesthesia.—Ethylene or vinyl ether U. S. P. (vinethene®) is used in the induction stage, followed by the administration of ether to a stage of proper relaxation, at which the anesthetic is maintained.

Technic of Tonsillectomy.—Sterile drapes are applied and the operation is conducted with the patient supine, with head hyperextended. A modified guillotine removal of the tonsils is made with a Beck instrument, the upper pole and pillars having first been freed by sharp and blunt dissection. The La Force adenotome is used for the adenoidectomy, Barnhill curets and punch forceps serving for the removal of small remnants. Careful hemostasis is secured in the tonsil fossae with use of clamp and suture. The assistant depresses the tongue and maintains suction in the pharynx. The concave head mirror and electric lamp are used for illumination.

Laryngoscopic Tracheobronchial Aspiration.—At the close of the operation, the Jackson laryngoscope is inserted and the larynx is exposed to direct vision. Through the open glottis, tracheobronchial aspiration is performed, a silk woven catheter being directed into the trachea and then into the major bronchi by manipulation of the head. Secretions removed by intermittent suction are collected in a 20 cc. calibrated bronchoscopic collection tube interposed into the suction system. A measured amount of isotonic saline solution is pulled through the suction apparatus after its removal from the tracheobronchial tree, and this amount is subtracted from the total reading on the calibrated collecting tube to give an accurate determination of the amount of bloody secretion aspirated. This method is useful in cases with scanty secretions. In such instances the bloody mucus is retained in the aspirating catheter unless washed into the collecting tube.

RESULTS

Of the 129 patients examined, 125 (96.9 per cent) had recoverable bloody secretion. Most of the patients were children below 12 years. Of those patients showing secretions, the greatest amount of secretion recovered from any single patient was 15 cc., and the least amount, 0.5 cc. However, 8 patients (6.4 per cent) showed 10 cc. or more, and 26 (20.8 per cent) showed 5 cc. or more. The over-all average was 3.7 cc.

Comment.—These findings coincide materially with those of other observers. Hara [17] performed bronchoscopy on 110 patients after tonsillectomy with general anesthesia, finding the presence of aspirated blood in each. Myerson,[15] in a similar series, showed that 76 patients out of 100 with tonsillectomy performed with general anesthesia had bloody secretion below the larynx. Iglauer [18] reported that bronchoscopy by

different observers on a total of 400 patients showed the presence of aspirated blood in 40 per cent of those patients on whom the tonsillectomy had been performed with general anesthesia and in 38 per cent of those, with local anesthesia.

PREVENTIVE MEASURES

Since it appears that blood easily finds its way into the tracheobronchial tree when tonsillectomy is performed with general anesthesia by the usual methods, a discussion of separate details of the operation will be offered.

Preoperative Medication and Anesthesia.—For children below 12 years, atropine is given hypodermically in a proper dose, 1/300 grain to 1/600 grain (0.2 mg. to 0.1 mg.), according to size and age, one hour before operation. For older children, morphine sulfate, ⅛ grain (8.0 mg.), may be given at the same time. Adults receive morphine sulfate, ⅙ grain (10.0 mg.), and scopolamine hydrobromide U. S. P., 1/200 grain (0.3 mg.), hypodermically an hour before operation.

This use of premedication conforms fairly well with methods in use generally, and it is thought that little improvement in technic could be realized from changes in doses, agents, etc. It is recognized that some anesthetists prefer the use of barbiturates in young children to allay fear and apprehension and lessen the "psychic trauma" of the trip to the operating room and anesthetic induction. The disadvantages of basal narcosis for tonsillectomy in children below 6 years, according to Kleinert,[21] are enumerated as increased induction time, administration of unnecessary drugs to children, the uncertainty of reaction of the drug and the shallowness of respiration and danger of laryngospasm, both of which are favorable to anoxia.

For adults, some anesthetists prefer atropine to scopolamine hydrobromide. A few anesthetists give no preoperative medication to children. The coming use of intravenous thiopental sodium (pentothal sodium®) and curare (*d*-tubocurarine chloride) for tonsillectomy in adults should be mentioned. While reports of its satisfactory use in tonsillectomy are multiplying, our experience with it has been limited.

Perhaps local anesthesia is the anesthesia of choice for tonsillectomy in the adult, but in cases in which a general anesthetic is desired, the use of the endotracheal tube with inflatable cuff is to be recommended, as it has not been found to hinder operative technic and helps to insure against aspiration of blood and pharyngeal secretions.

Ether remains the anesthetic of choice for children and is best given by the open method to a light surgical plane.

Position.—In the true supine position, the course of the trachea is posterior, bringing gravity to bear on the passage of pharyngeal secretions

21. Kleinert, M. N.: Premedication in Tonsillectomies in Children Under Six Years Old, Ann. Otol., Rhin. & Laryng. **56**:168-174 (March) 1947.

to the bronchial tree. While a downward 45 degree tilt (from the horizontal) of the table head permits satisfactory protection from aspiration, it forms an awkward operating position for the surgeon. It also predisposes to atelectasis as a result of elevation of the diaphragm and reduction of vital capacity. Shallow respiratory efforts are thereby produced, which are favorable to anoxia and fatigue of the respiratory center, as shown by Case and Stiles [22] and others.

A lesser degree of tilt, therefore, would seem more satisfactory, 10 to 15 degrees from the horizontal, affording a tracheal position not favoring gravitation of pharyngeal secretions to the lower air passages and not producing sufficient elevation of the diaphragm to reduce materially vital capacity. In addition, surgical procedure in such a position is not made difficult.

Of greater importance is the position of the patient's head, for when tonsillectomy is performed even with an adequate degree of tilting of the table, blood may enter the trachea in considerable amount if the head remains in a normal relation to the body axis. As shown by Dutton,[23] hyperextension of the head on the neck, in a modified Rose position, allows pooling of secretions in the nasopharynx, where they may easily be removed by suction.

An operating position combining slight downward tilting of the head of the table (10 to 15 degrees) and hyperextension of the head would appear to offer maximum protection from aspiration of pharyngeal contents during tonsillectomy with the patient under general anesthesia. It is necessary for the anesthetist or other assistant stationed at the head of the table to see that such positions are maintained at all times.

It was of interest in our own series to note a considerable reduction in the amount of aspirated secretions recovered by suction in the last 67 cases, in which more rigid adherence to proper positioning was followed as outlined here. Whereas in the first half of the series an average amount of recovered secretion was 4.6 cc., with several high individual readings over 10 cc., including two at 15 cc., the last half of the series showed an average of 2.7 cc., with the highest single individual amount at 6.5 cc.

The Davis-Crowe mouth gag, used by certain operators, permits a sitting posture by the surgeon at the head of the table and utilizes body and head positions in conformity with those previously outlined. A disadvantage voiced by some operators is that complete removal of the fringe of adenoid at the junction of the nose and nasopharynx is made difficult when the tongue spatula is in place. Some surgeons there-

22. Case, E. H., and Stiles, J. A.: The Effect of Various Surgical Positions on Vital Capacity, Anesthesiology **7**:29-31 (Jan.) 1946.

23. Dutton, A. C.: Posture During Anesthesia—Its Effect, California & West Med. **37**:145-150 (Sept.) 1932.

fore perform adenoidectomy by conventional methods, then insert the Davis-Crowe mouth gag and proceed with the tonsillectomy. Shambaugh,[24] on the other hand, has devised a special "reversed curve" modification of the La Force adenotome to facilitate adenoidectomy with the Davis-Crowe gag in place.

Operative Technic.—Adequate illumination of the pharynx is a necessity. The concave head mirror or the focusing head lamp worn by the operator are the instruments of choice for adequate illumination. Adenoidectomy is best performed first so that bleeding may cease during tonsillectomy. Complete removal of the adenoids by adenotome, curet and biting forceps should be made. The Love palate retractor and the Yankauer nasopharyngeal speculum are valuable instruments for examination of the nasopharynx. Rosenmüller's fossae should receive particular attention.

Any technic of tonsillectomy is satisfactory which leaves pillars and capsule intact and accomplishes removal of the plica semilunaris and plica triangularis. Suction must be used during this part of the operation as well as during adenoidectomy to prevent overflow of secretions into the larynx. Suction is most efficiently performed by an assistant, when the operator assumes his usual position at the side of the patient's head. Even so, some spillage may occur. Careful hemostasis, with clamp and ligature or suture for bleeding vessels, will materially reduce the amount of blood entering the pharynx during operation and also during the immediate postoperative period.

The patient is returned to the room with the head turned to one side, in a semiprone position, with shoulders prone but hips in a lateral position elevated on a small pillow. This position is maintained by the upper knee being flexed and drawn up; it is held until the patient has reacted from the anesthetic. Such a positon favors postural drainage, yet does not reduce vital capacity owing to elevation of the diaphragm. Likewise, the venous pressure is not so markedly increased as with severer head-low positions. There is less tendency, therefore, for postoperative bleeding. Deep breathing and frequent turning are encouraged.

At the conclusion of the operation, after hemostasis has been secured and all tampons removed, direct laryngoscopy is done and aspiration of the tracheobronchial tree is carried out.

Laryngoscopic Tracheobronchial Aspiration.—In the past, many methods for removing aspirated blood have been used. Some surgeons allow the patient to remove it by coughing. Years ago, tonsils and adenoids were removed swiftly and the patient was immediately placed in a prone position with head hanging over the end of the table until bleeding subsided and aspirated blood was apparently coughed up. The

24. Shambaugh, G. E., Jr.: Diseases of the Pharynx and Fauces, in Jackson and Jackson,[14] p. 159.

head-low position, a form of postural drainage, has been used, the blood being removed from the pharynx by suction. Manual compression of the thorax to force blood into the pharynx is another method.

We believe the ideal method is that which combines proper anesthesia, positioning as previously outlined, careful operative technic and hemostasis, the use of adequate pharyngeal suction and direct laryngoscopy with tracheobronchial aspiration at the close of operation. The silk woven catheter attached to the suction tubing is atraumatic, yet stiff enough to be easily manipulated. In sizes 10 to 18 French, a range for all cases is assured. By manipulation of the head from side to side, the catheter can be caused to enter either main bronchus. The stimulating effect of the catheter on bronchial reflexes produces the so-called tussive squeeze, which clears aspirated material from the segmental branch bronchi, bringing it up to a level accessible to suction. An extra step in the prophylaxis of postoperative atelectasis, bronchopneumonia and pulmonary abscess has therefore been taken. A definite reduction in the amount of postoperative coughing and vomiting is noted in patients treated by aspiration of the tracheobronchial tree. This reduction is of advantage, since these acts raise the venous pressure and are favorable to bleeding in the postoperative period. A reduction in the postoperative morbidity is thereby possible.

The method has been used to give training in the use of the laryngoscope to residents and others. Otolaryngologists have found the routine use of laryngoscopic tracheobronchial aspiration after tonsillectomy a valuable means of becoming more facile with the laryngoscope and of refreshing their memory of the normal anatomy of the child's larynx.

SUMMARY

1. Tonsillectomy is probably the most frequently performed surgical procedure in the United States.

2. Atelectasis and pulmonary abscess not infrequently complicate tonsillectomy performed with general anesthesia.

3. Aspiration of pharyngeal contents during tonsillectomy is favorable to pulmonary complications.

4. The method is outlined of performing laryngoscopic tracheobronchial suction immediately after operation.

5. In a series of 129 patients on whom tonsillectomy was performed with general anesthesia, 125 (96.9 per cent) were found to have measurable amounts of bloody secretion in the tracheobronchial tree.

6. Measures are presented designed to minimize the possibility of aspiration during tonsillectomy and adenoidectomy with the patient under general anesthesia.

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SELECTION OF PATIENTS FOR FENESTRATION SURGERY

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SELECTION of patients for fenestration surgery is entirely dependent on the ability to recognize certain characteristic features which, in combination, comprise the clinical entity of otosclerosis. As in certain other labyrinthine disturbances, the correlation between the pathologic picture and the clinical manifestations thereof is a matter of presumption based on retrospection in the autopsy rooms, in the histology laboratory and on observations made at the operating table.

Otosclerosis is a histologic diagnosis. The principal structures involved are the oval and round windows and the cochlea. The presence of otosclerosis may be determined with certainty only when the temporal bone is made available for microscopic study. Apparently this condition occurs only in man and has never been reproduced identically in animals, although considerable effort to do so has been made.[1]

For more than half a century otosclerosis has been and still is the subject of intensive research and not a little philosophic reflection. While attention has been directed to the possible biologic, metabolic and vascular aspects suggested by certain peculiarities of the histolgic picture, the present state of elucidation takes one but a short way from the periphery to the center of the problem concerning its cause.[2] Most of these aspects have been investigated intensively and repeatedly, yet the solution is no nearer now than when Politzer first described the condition as a disease entity.[3] His conception has since diminished in favor, and in its stead mounting evidence is accumulating which tends

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1. Weber, M.: The Bone Picture of Otosclerosis and the Theory of Its Experimental Reproduction, Tr. Am. Otol. Soc. **20**:55-99, 1930.

2. Fowler, E. P.: Calcium, Phosphorus and Cholesterol in Otosclerosis, Tr. Am. Otol. Soc. **20**:39-47, 1930. Guggenheim, L. K.: Otosclerosis, St. Louis, The Author, 1935, pp. 1-54.

3. Politzer, A.: Text-Book of the Diseases of the Ear and Adjacent Organs, for Students and Practitioners, translated and edited by J. P. Cassells, Philadelphia, H. C. Lea's Son, 1883, pp. 324-353.

to shift attention to the consideration of vascular phenomena.[4] This consideration appears to be more attractive, but it is still inconclusive. The cause for such vascular alterations is still to be determined. While the etiologic picture has remained obscure, remarkable progress has been made in developing surgical technics, mechanical equipment and electrical devices which offer the afflicted persons a chance to escape solitary confinement in the dismal atmosphere of deafness. How much this has meant to the problem of cracking the hard shell of occult circumstances surrounding the development and course of otosclerosis is shown by the renewed interest of investigators intent on taking the utmost advantage of the information furnished by operative procedures on the temporal bone.

Although the results of fenestration surgery are as phenomenal as the possibilities for future investigation opened up by it, there still remain the more immediate and practical problems of accurate diagnosis and selection of patients for surgical treatment. Some of the clinical manifestations of otosclerosis are relatively easy to recognize. They form a well known pattern, which defines only in part the term "clinical otosclerosis." According to common but indiscriminate habit, this term is often used for the singular purpose of ascribing suitability of a patient for operation.

Just what range or scope of physical and functional features of hearing loss clinical otosclerosis actually should embrace is not precisely known, but it is obvious that it includes, at least, certain characteristic findings which serve as criteria for selection of patients who may be offered fenestration surgery. The term clinical otosclerosis is generally thought to describe a hearing loss of the type in which the bone conduction hearing in audiometry is approximately normal and considerably better than the air conduction threshold, which is elevated without demonstrable clinical cause. There has been considerable speculation in favor of the argument that otosclerosis may be responsible for certain instances of so-called nerve deafness as well, the point being raised because the term "clinical otosclerosis does not necessarily imply that the hearing loss may be corrected by or is amenable to operation, even though the indications are that the underlying pathologic condition is otosclerosis. Hence, the term clinical otosclerosis should include, but not be limited to, those clinical patterns which may be expected to respond favorably to operation.

Since the histologic and the clinical evidence cannot be correlated simultaneously, it is obvious that this difficulty constitutes the principal deficiency in the problem of diagnosis. However, it is equally obvious

4. (a) Wittmaack, K.: The Pathogenesis of Otosclerosis, Arch. Otolaryng. **14**:186-194 (Aug.) 1931. (*b*) Lempert, J., and Wolff, D.: Otosclerosis: Theory of Its Origin and Development, ibid. **50**:115-155 (Aug.) 1949.

that accuracy of clinical diagnosis is firmly supported by the high percentage of initial surgical success. The well known fact that subsequent failure does occur in no way indicates an error in proper and accurate selection.

The problem of selection is relatively simple when the patient under consideration demonstrates the following clinical features:

1. Bilateral hearing loss in which air conduction thresholds exceed approximately normal bone conduction thresholds by 30 decibels or more
2. Tympanic membranes are normal in appearance
3. Roentgenograms of the temporal bones show no evidence of residual infection
4. Hearing is good with use of the telephone or a hearing aid and seems to be better in a noisy environment
5. Onset of loss of hearing occurs after pubescence and is not related to systemic or local infection
6. The hearing loss is alleged to have progressed and then to have remained stationary or to have continued to progress slowly
7. Blood relatives of the same or antecedent generations have or have had a similar hearing loss
8. Tinnitus and/or vertigo expressed as current or past complaint

Except for the characteristic pattern of hearing loss as determined by bone and air conduction thresholds, the omission or variation of one or more of the criteria listed here need not constitute cause for rejection.

EXTRACTS FROM CLINICAL HISTORIES

According to these associated factors, it becomes apparent that the three essential clinical sources from which accuracy in selection develop are history, physical examination and hearing tests.

The patient who presents the classic picture of clinical otosclerosis which is amenable to surgical restoration of practical hearing possesses a hearing loss which can be effectively improved by sufficient amplification. Hearing for speech is usually better in an environment of relatively high sound intensity and is quite acute when the source of sound is in the immediate proximity of the ear. These facts may be determined by the alert examiner during the objective examination procedures, without recourse to the unreliable and unwarranted, by conventional spoken or whispered voice tests.[5] The otologist, experienced and basically trained in the problems concerning the diagnosis of clinical otosclerosis, has little difficulty in reaching an accurate conclusion regarding the eligibility of the patient for operation and predicting the prospects of a successful result. The degree of success to be expected parallels very closely the audiometric features of the hearing loss. Audiometry is essentially

5. Glorig, A.: Hearing Evaluation by Low Conversational Voice Tests, Ann. Otol., Rhin. & Laryng. **58**:394-402 (June) 1949.

the pivot around which all other features of clinical otosclerosis revolve, and these features are assessed according to their related significance. They may be regarded as confirmatory aids in reaching the logical diagnosis.

The hereditary nature of the condition is recognized as a significant element of otosclerosis, and numerous affected families have been investigated, but a positive history of familial deafness may not always be obtained.[6] This situation should not be accepted to mean that no such history exists and should never discourage the diagnosis indicated by other factors. Apparently otosclerosis may exist in the line of descendancy for generations before becoming manifest, it being four times as prevalent among women as among men. In relatively few instances the family history is unknown to the subject because ancestral contacts have been severed or lost. Information on familial deafness sometimes is withheld for security reasons against the stigma of a hereditary malady or because, for some obscure purpose, the patient prefers not to divulge it. Not infrequently familial history of clinical otosclerosis is rather distant from direct descendancy. Whether such tenuous evidence of ancestral involvement is significant may be doubtful but is suggestive. Nevertheless, it is an established fact that many patients who deny family history of deafness are counted among those who have had their hearing restored to practical serviceable levels after fenestration surgery.

The temperament of patients with inadequate hearing differs somewhat according to the type of hearing loss, age, occupation and sex. These factors are powerful influences which may be brought to bear on both the otologist and patient in reaching a mutual agreement on the choice of therapy. The consultant's decision and advice are dictated by the possibilities presented in the results of his examination. The patient's motivation in seeking relief varies from social inadequacy to economic insecurity. Less obvious psychologic pressures not directly related to these may explain why a determined but irresponsible patient may wish to force the surgeon's hand against rational concepts of selection. Whatever the motivations, the hard of hearing patient will grasp tenaciously at any opportunity which offers even the slightest hope of deliverance from bondage.

It is not within the scope of this paper to discuss details pertaining to these subjects, but it should be emphasized that psychiatric consultation can be of considerable help in avoiding some of the undesirable repercussions resulting from surgical events, even if the operation is successful. It is also to be kept in mind that psychologic aberrations may establish firm foundations among the mental stresses and strains

6. Davenport, C. B.: Hearing in Children When Both Parents Have Otosclerosis, Tr. Am. Otol. Soc. **20**:48-54, 1930.

of deafness and that the liberation of the patient from a more or less isolated existence in the world of sound may easily change the entire clinical panorama.

Pregnancy has been suspected of precipitating or aggravating hearing loss due to otosclerosis. It must be remembered that clinical otosclerosis usually makes its appearance during the child-bearing span of life and that the association of hearing loss and pregnancy may simply be a coincidence. It is this frequently observed phenomenon which has stimulated so much effort to find the cause of otosclerosis in the processes of metabolism and endocrine activities. Further elucidation must await more complete and accurate information concerning this subject.

The age at which onset of deafness due to otosclerosis is noted usually coincides with that period of development occurring with or following pubescence. However, in some instances in which the events of anamnesis are recalled vaguely the patient may assert that the time of onset is much earlier or much later.

It is helpful to know when the hearing loss first became noticeable and when it became a definite handicap. This information gives some indication about the rate of progress and the prognosis. Usually, the earlier the onset, viewed with the extent of hearing loss at the time of examination, the more doubtful one can be of a favorable prognosis regarding the longevity of adequate cochlear reserve. Whether or not fenestration surgery influences the rate of secondary neural degeneration is still doubtful. It is likely that neural depression will continue to follow its destined course regardless of a successful fenestration operation, but during that period of serviceably functional retention the patient may enjoy whatever improvement the cochlear reserve will permit.

Some have asserted that younger clinical otosclerotic patients stand a better chance of improvement in hearing than older patients. If it is meant that incidence of operability is greater among young persons with otosclerosis, concurrence with such views is acceptable. But if it is meant that failure is more frequent among elderly patients who have been carefully selected and operated on, then such opinions are in contrast to the studies so far conducted at the University Hospitals. The reason for this difference of opinion is not entirely speculative, although the subject could stand considerably more investigation. Failure subsequent to fenestration surgery seldom occurs among elderly patients who have excellent hearing for bone-conducted sounds. It may be inferred that such patients possess adequate cochlear function and that the hearing improvement which they experience as the result of fenestration surgery is not so likely to deteriorate as rapidly as an early presbycusis would suggest. Osteogenetic closure of the fenestra is more remote among those over 35 years of age.

An age limit alone as a basis for admission or rejection of patients for consideration of fenestration surgery cannot be unalterably defined. This factor is subject to individualized treatment. The natural processes of growth, development and degeneration control the circumstances in which prudence and judgment must be exercised. Rather than be guided by an arbitrary age limit, some effort in thought on the part of the assessor is required. The operation is certainly not dangerous to the healthy person, and the fact that a remarkable record has been sustained to support such a statement is ample testimony to the care that has been exercised in this regard.

Tinnitus is one of the disagreeable symptoms usually associated with clinical otosclerosis. During the progressive phases of the hearing depreciation particularly it is usually described as of low-pitched, roaring or pulsating quality. It may be inferred according to the audiometric results that complete stapedial fixation has not yet occurred. Many patients who declare their hearing loss to have been stationary for years seldom complain of tinnitus. Those who do usually describe the tinnitus as high-pitched ringing or whistling sounds. Audiometric evidence of neural degeneration may frequently be the accompanying clinical feature of this complaint.

Vertigo is rarely a complaint among young patients, but it is not infrequent among elderly persons with clinical otosclerosis. It is rarely incapacitating, but at the same time it is seldom completely relieved and is occasionally exaggerated after the fenestration operation. One should be cautious in leading the patient to believe that such a symptom will diminish or disappear as the result of the operation.

OBJECTIVE APPEARANCES

Some physical characteristics of the external auditory canal and tympanic membrane noted by early writers are found in association with clinical otosclerosis. Frequently the meatus and canal are exceptionally spacious and devoid of the usual ceruminous and epithelial debris found in the average auditory canal. The appearance of atrophy of the skin of the canal and the drum membrane, which is often noted, may be explained by the theory of vascular alterations attributed to Gray [7] and to Holmgren.[8] The sensitivity of the auditory canal to tactile stimulation is reported to be diminished in clinical otosclerosis. It is questionable at times that such is the case.[9]

7. Gray, A. A.: The Otosclerosis Problem, Including Reports of Two Cases Examined Pathologically, J. Laryng. & Otol. **49**:629-663 (Oct.) 1934.

8. Holmgren, G.: Surgical Therapy in Otosclerosis, in Loose-Leaf Surgery of the Ear, New York, Thos. Nelson & Sons, 1947, pp. 399-430.

9. Wojniak, F.: The External Ear and Drum Membrane in Otosclerosis, Ann. Otol., Rhin. & Laryng. **55**:406-419 (June) 1946.

Quiescent external otitis may constitute a serious hazard to the successful outcome of the fenestration operation. Even though the epithelial lining may appear grossly normal, a history of recurrent itching and burning of the ears is not to be taken lightly. The restricted vascular supply and the perivasculitis which pathologically defines this condition may flare at the instigation of surgical trauma.[10] The result is likely to terminate in a devitalized tympanomeatal membrane, with ensuing failure of improvement in hearing.

It is unfortunate that occasionally clinical otosclerosis may be somewhat obscured by evidence of past inflammation of the tympanic membrane. So far as is known, otosclerotic ears are no more immune to inflammatory attacks than ears with normal hearing. Politzer's dilemma seems to have been reversed in our time. He found it difficult to explain middle ear deafness in normal ears, which he nevertheless labeled chronic catarrhal otitis media.[8] Now there appears to be some reluctance to ascribe the diagnosis of clinical otosclerosis to ears which even slightly suggest evidence of residual infection. Lempert drew attention to the clinical variations that might be encountered during the process of hearing assessment which would not contraindicate the diagnosis of clinical otosclerosis.[11] Within bounds of logic, it is to be expected that the patient exhibiting clinical otosclerosis should sustain the scars of aural infection occurring in the span of life preceding the detection of the hearing deficiency. How many case records exist filed under the diagnosis of chronic adhesive or catarrhal deafness may be determined only by a critical review of the facts in each case, but there is some indication of the numbers of patients so labeled who subsequently enjoy restoration of practical hearing as the result of fenestration surgery.

There cannot be general agreement with those who contend that the operation is being performed unintentionally on patients with obstructive deafness other than otosclerosis. No doubt this situation has occurred, and probably will continue to occur, in those instances in which certain facts remain ignored. However, attention is invited to a review of some of these considerations.

Fixation of the ossicular chain may occur at different anatomic locations: (1) the drum membrane; (2) incudomalleal joint; (3) incudostapedial joint, and (4) the stapedial footplate.

The various possible causes have been enumerated in standard textbooks, and we will not consider them here except to point out that ossicular fixation occurring elsewhere than at the stapedial footplate is a rare experience among those who have investigated such possibilities in oto-

10. Kos, C. M.: Evaluation of the Diagnosis and Treatment of External Otitis, Iowa State M. J. **39**:560-566 (Dec.) 1949.

11. Lempert, J.: Lempert Fenestra Nov-Ovalis with Mobile Stopple, Arch. Otolaryng. **41**:1-41 (Jan.) 1945.

sclerosis, even though abnormal changes are to be found microscopically.[4b] It is a tribute to good judgment and the ability to appraise accurately the observations made prior to and at the time of exposure of the ossicular chain that such a possibility remains so remote. There has been no convincing evidence from personal experience or from a search of the literature that the creation of a fenestra favorably influences hearing if the stapes remains freely mobile.

If the diagnosis is found to be erroneously based at the time the ossicular chain is exposed, the fenestration stage of the operation should be omitted. Immobilization of the ossicular chain other than at the stapedial footplate is obviously corrected the moment the incus is dislocated and extracted. Hence, the fixation being released, there is restored ample allowance for movement of the stapes and a resultant improvement in hearing, which can hardly be exceeded by the fenestrated ears in which the stapes is fixed. The following abstract of a patient's record is an example.

REPORT OF A CASE

J. C., a white male student at the university, was unaware of any impairment of hearing until June 1946, at which time he noted that it was necessary for him to ask that words and sentences directed to him be repeated. However, he ignored the possible implications such experiences indicated to him until he was examined for acceptance into a course for communications skills. He denied any history of aural infection or otalgia, and there was no information given suggesting a family history of deafness. His nose, nasopharynx, pharynx and larynx were normal. The paranasal sinuses were perfectly clear on roentgenographic examination. The periauricular tissues appeared normal, as did the external auditory canals. The drum membranes were translucent and revealed no evidence of past inflammatory involvement.

The roentgenograms of the mastoid processes were read as showing "densely sclerotic bone with underdevelopment of the cells. The mastoid tips are both diploic in nature." Vestibular function was normal according to caloric and rotational tests. Audiograms and results with tuning fork tests were commensurate with the diagnosis of clinical otosclerosis excellently disposed for fenestration surgery (chart 1 *A*).

The fact that the mastoid processes were not clearly pneumatic in appearance roentgenographically did not obviate the clinical diagnosis, inasmuch as this laboratory finding is occasionally observed in otosclerotic patients who are acceptable for fenestration surgery. After study and review of the clinical features presented on frequent occasions over a period of more than a year, an operation was performed with the intention of proceeding with the creation of a fenestra nov-ovalis. With reference to the operative record, the following notations were made:

"It was noted that the mastoid process was semisclerotic and that the sinodural cells were filled with a noninflammatory material of lipid character, as was the retrofacial tract of cells. A rather dense membrane approximately 0.5 mm. in thickness partitioned off the aditus and attic of the middle ear from the antrum and mastoid cells. There was no evidence of active inflammation. (The pathologic report later read: 'chronic inflammatory tissue.') A curtain of membrane, almost transparent, surrounded the attic projections of the ossicular chain. The incus

was rather firmly attached to the head of the malleus. The incudostapedial joint was exposed and the stapes tested for mobility. It appeared to be fixed. However, when the incus was removed, the stapes appeared to become mobile when tested with the paracentesis knife. The remainder of the middle ear cavity was normal in every respect."

As the result of these observations the fenestration stage of the operation was omitted. The tympanomeatal membrane, having been fashioned, was fitted into position covering the semicircular canals, and the operation terminated with the placement of the dressings. The first postoperative audiogram was made on Jan. 17, 1949 (chart 1 *B*). The last audiogram, taken on June 7, revealed a remarkable improvement in hearing (chart 1 *C*).

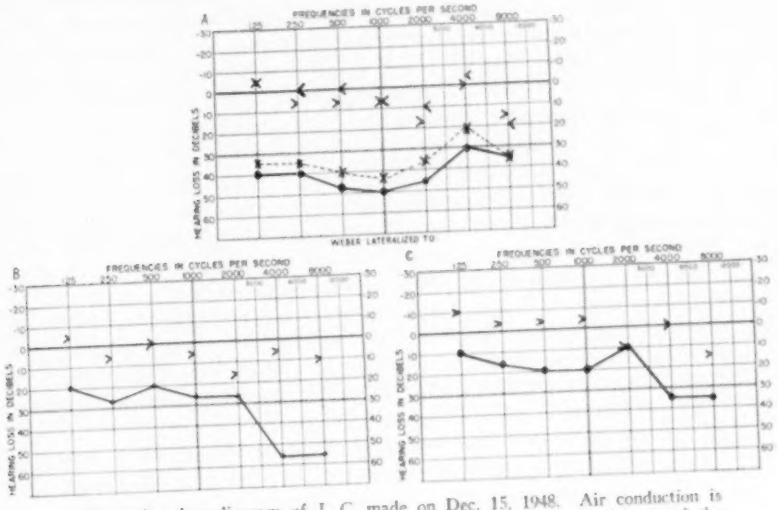

Chart 1.—*A*, audiogram of J. C. made on Dec. 15, 1948. Air conduction is indicated by crosses; bone conduction, by arrowheads. Arrowheads toward the left indicate the left ear; toward the right, the right ear. Ossicular fixation at the incudomalleal joint was discovered at operation. Preoperative diagnosis was clinical otosclerosis. *B*, made on Jan. 17, 1949, after removal of incus and head of malleus. Arrowhead indicates bone conduction of the right ear. *C*, made on June 7, six months after operation. The cavity was completely epithelized and dry. Arrowheads indicate right ear.

Stapedial fixation has been generally confirmed by relatively crude technics, which consist of placing a probe on the head of the stapes to test its freedom of mobility. It is possible by such methods to break, with only the slightest pressure, tenuous otosclerotic bone holding the stapes in place. Occasionally the crura fracture leaves the firmly fixed footplate undisturbed. Such alterations incurred by the usual methods of testing for stapedial fixation, after the incus has been removed, may account for

the lack of certainty in reporting complete stapedial fixation in every case with a preoperative diagnosis of clinical otosclerosis. In addition, it is probably not necessary that the stapedial fixation be complete to assure a beneficial operative result, all other factors entailed in successful surgical procedure being considered. While such experiences are uncommon, we are sure that others as well as we have encountered instances of apparently detectable stapedial mobility with most favorable response to fenestration surgery.

The eustachian tube has been found to be normally inflatable with such regularity in clinical otosclerosis that it is extremely doubtful that this test really lends much support to the diagnosis if the drum membrane appears normal. The hearing loss, the picture of the drum membrane and tubal function invariably coincide as correlated influences affecting the clinical picture. In uncomplicated clinical otosclerosis the hearing threshold is never appreciably altered after inflation. In questionable instances, however, adequate patency should be determined and the effect on subsequent audiometry should be noted.

After the elements of distress presented by the patient have been ascertained, attention is directed to the responses shown by the auditory organs to various sources of sound stimulation. A battery of tests are employed to determine the liabilities and the assets of the auditory system. Responses to speech and pure tones under varying test conditions serve to augment the initial impression obtained during the preliminary physical examination. The tuning forks, in experienced hands, reveal invaluable information to support other carefully conducted tests or to contradict technical carelessness in adjunctive testing. Audiometry, as obtained by the pure tone and speech audiometer, is capable of producing more extensive and more accurate data, but the reliability of the results is largely dependent on the examiner who uses the equipment.

EVALUATION OF FUNCTIONAL HEARING TESTS

If the history and physical examination impose no contraindication, the results of the functional hearing tests not only determine the advisability of surgery but also indicate in advance the probability of the excellence of postoperative hearing acuity.

As the result of accumulated experience, certain general criteria for the selection of cases and evaluation of the end result as failure or success are widely accepted. For example, the patient who has a bilateral air conduction loss of 30 decibels or more associated with normal or better than normal hearing for bone-conducted tones, as generated by the audiometer over the frequency range of 512, 1024, and 2048 cycles, is most likely to obtain a successful surgical result. And unless operation

results in an increase of the average air conduction acuity at 512, 1024 and 2048 cycles to the 30 decibel critical level or better, it may be considered unsuccessful, even though a considerable and appreciable gain ensues. Such gains, while not completely satisfactory, enable some patients to obtain improved assistance with a hearing aid. Any result short of these standards renders the fenestration procedure doubtful in value.

Most otologists agree that the excellence of the patient's bone conduction acuity is of utmost significance in the process of selection for fenestration surgery. All other factors being considered, the patient with an air conduction hearing loss of 30 decibels or more and normal bone conduction acuity theoretically should obtain a better end result than the patient with identical or even greater air conduction loss but with subnormal bone conduction acuity.

While there exists a divergence of opinion concerning the reliability of measurements for bone conduction acuity as criteria for selection,[12] there is ample reason to believe that the prefenestration bone conduction acuity constitutes an exceedingly significant standard of selection. It should be pointed out that measurements to which reference will be made in succeeding statements were obtained under laboratory research conditions by one whose experience spans about twenty years of testing hearing in both clinical and research circumstances. The bone conduction vibrator used in obtaining measurements of the bone conduction acuity is a precision instrument carefully calibrated on young adults with normal air conduction acuity in an exceedingly quiet room. The vibrator is mounted in a spring headband so that it is held with approximately the same pressure against each patient's mastoid process. A more complete description of the bone conduction receiver and its calibration is contained in an article by Lierle and Reger.[13]

In order to ascertain the value of the pure tone auditory acuity in relation to selection criteria, a series of 47 consecutive case records of patients were studied. Presurgical hearing thresholds represent the averages of three tests each of air and bone conduction acuity. The postfenestration hearing levels were obtained by averaging the three best (nearest normal) hearing tests of each patient after his acuity had reached its apparent permanent level. Although both air and bone conduction acuity measurements were obtained at octave intervals from 64

12. Walsh, T. E., and Silverman, S. R. P.: Diagnosis and Evaluation of Fenestration, Laryngoscope **56**:536-555 (Sept.) 1946.

13. Lierle, D. M., and Reger, S. N.: Correlations Between Bone and Air Conduction Acuity Measurements over Wide Frequency Ranges in Different Types of Hearing Impairment, Laryngoscope **56**:187-224 (May) 1946.

to 8192 cycles inclusive, only the results at 512, 1024 and 2048 cycles are considered here (table).

Since these data appear to bear a definite relation to the accuracy of selection for fenestration surgery, they are classified in order to explain more fully their significance. For example, inspection of the first category shows the preoperative bone conduction acuity and the postoperative air conduction acuity of the 15 patients who sustained the best postoperative results, which were an average air conduction acuity of 20 decibels or better at 512, 1024 and 2048 cycles. The average preoperative bone conduction acuity of the 15 patients at 512 cycles is 2.4 decibels. This figure is based on three separate tests of each patient, making a total of 45 measurements at the 512 frequency level. The preoperative bone conduction measurements shown at 1024 and 2048 cycles were obtained in a similar manner. With illustrative graphic plotting of these data with the *x* and *y* axes (abscissa and ordinate), as employed

Preoperative Bone Conduction and Postoperative Air Conduction at 512, 1024 and 2048 Cycles Per Second

	Preoperative Bone Conduction by Decibels Cycles			Postoperative Air Conduction by Decibels, Averages at 512, 1024 and 2048 Cycles
No. of Cases	512	1024	2048	
15	−2.4	−0.6	9.8	19.9 or less
14	−3.5	1.0	14.1	20-24.9
9	2.7	6.9	20.8	25-29.9
4	6.9	11.0	26.9	30-34.9
5	7.5	13.0	32.5	35 or more

in the construction of the conventional audiogram blank, it becomes apparent that there is a consistent relation between the excellence of the preoperative bone and postoperative air conduction acuity (chart 2).

Chart 3 summarizes the preoperative averages of bone conduction acuity at 512, 1024 and 2048 cycles and the postoperative air conduction averages for the same frequencies for the 47 patients. These data are presented as a curve in which the average preoperative bone conduction is plotted against the postoperative air conduction.

In addition, chart 3 includes a scattergram which shows the exact average of preoperative bone and postoperative air conduction acuity of each of the 47 patients. For example, the encircled dot on the 5 decibel ordinate indicates that this patient had an average preoperative bone conduction acuity of 5 decibels and obtained a postoperative average air conduction acuity of 18.3 decibels; the encircled dot on the extreme right of the chart indicates that this patient had a preoperative bone conduction acuity of 9.1 decibels and a postoperative air conduction acuity of 52.7 decibels, indicating a failure in this series of cases.

The slope of this unsmoothed curve indicates a systematic relation between the preoperative bone and postoperative air conduction acuity. The positions of the dots (cases) relative to the curve show the magnitude of the individual deviations from the average. Even though the curve is constructed on data from only 47 patients, its slope and curva-

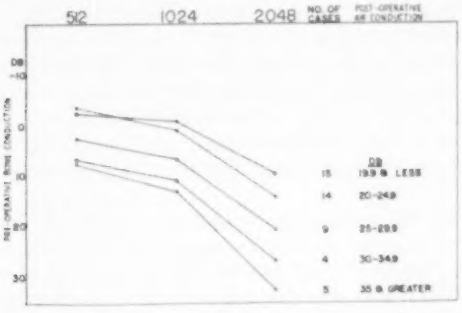

Chart 2.—Preoperative bone conduction and postoperative air conduction at 512, 1024 and 2048 cycles per second.

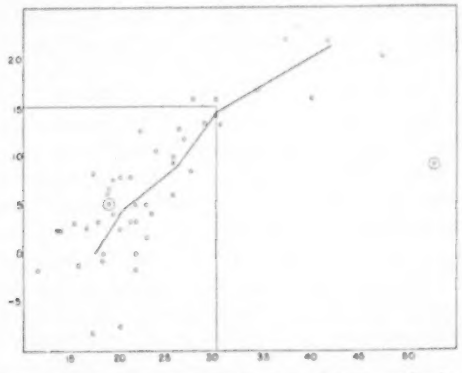

Chart 3.—Average postoperative air and bone conduction.

ture are sufficiently systematic to serve as a useful guide in predicting the postoperative result from the preoperative bone conduction measurements. For example, if the average preoperative bone conduction acuity is 5 decibels, the curve indicates that the average postoperative air conduction acuity will be approximately 21 decibels. The curve also indicates that an average preoperative bone conduction loss of 15 decibels will result in an average postoperative air conduction acuity of 30 deci-

bels and that an average preoperative bone conduction loss greater than 15 decibels probably will result in an average air conduction loss in excess of 30 decibels. Predictability may even be extended beyond these critical limits. It will be noted that of the 47 patients considered, 40 possessed preoperative bone conduction acuity levels of 15 decibels or less at the 512, 1024 and 2048 frequencies. And of these 40 patients, 39, or 97.5 per cent, had postoperative air conduction acuity of 30 decibels or less.

It must be emphasized that these curves are based on data from only 47 subjects and that the predictability of postoperative results based on the preoperative bone conduction acuity should be regarded as approximations only. However, the acquisition of additional data will permit the construction of a predictability curve of known statistical probability.

Inasmuch as the postoperative results in these 47 patients were obtained with a given surgical technic, it may be expected that in the event it is considered desirable to vary the surgical management or procedure, the results would be reflected by comparison in the method illustrated for plotting preoperative bone conduction acuity against the postoperative air conduction thresholds.

As a consequence of these studies, it is believed that the hearing thresholds of clinically otosclerotic patients may be evaluated according to three distinct audiometric patterns. For the purpose of having a more or less fixed protocol for selection, the following classification is useful:

Class 1. Bone conduction acuity does not exceed an average loss of 15 decibels at 512, 1024 and 2048 cycles, and the intensity differential described as the difference between air conduction and bone conduction thresholds is at least 30 decibels—preferably more. If the bone conduction threshold remains within normal limits, the intensity differential rarely exceeds 60 decibels in carefully tested ears.

Class 2. Bone conduction acuity does not exceed an average loss of 20 decibels, and the intensity differential is at least 30 decibels at 512, 1024 and 2048 cycles.

Class 3. Bone conduction acuity exceeds average loss of 20 decibels at 512, 1024 and 2048 cycles.

The pure tone audiometric criteria for determining the suitability of the patient for fenestration surgery are set forth according to an analysis of preoperative and postoperative data obtained through personal experience and from comparisons made with published reports dealing with this subject. Perhaps the limitations applied are too stringent in some instances, but there is a higher degree of reliable predictability for this classification than if it were to be relaxed in any respect.

In accordance with the standards set forth for class 1, it will be noted that any extension of the limitations prescribed might very seriously compromise a successful initial postoperative hearing improvement in as many as 97 per cent of patients. With few exceptions, those who did not meet the minimum requirements failed to fulfil the expectation of reaching an improved hearing level of 30 decibels for the conversational frequencies. Hence, we consider the average preoperative bone conduction threshold of 15 decibels to be that critical point from which deviation is likely to culminate in disappointment for both surgeon and patient.

Depending on individual considerations, those in class 2 may be given some encouragement as to the possible merits of fenestration surgery, but it is to be emphasized that favorable expectations of achieving improvement in hearing to the practical serviceable level of 30 decibels are very remote.

With very few exceptions, those who fall into class 3 had better not be selected for fenestration surgery. Despite a technical success, very little appreciable hearing improvement is subjectively experienced. These patients are apt to float for awhile on the carpet of wishful thinking. Their buoyancy carries them and temporarily sustains them at imaginary levels. They delude themselves, as they always have done since the onset of their hearing loss, until some resented but forceful and embarrassing incident or series of experiences awaken them to the sober facts. Only then do they reconcile themselves to using the hearing aid again.

SELECTION OF PATIENTS FOR REVISION

Since a certain number of patients fail to retain permanent hearing improvement after the fenestration operation, the problem of selection for revision occurs for careful consideration. It has been pointed out in a classic discussion of this subject that, accidents excluded, osteogenesis, fibrosis and labyrinthitis, in this sequence of importance, are the causes of postoperative failures.[14] Assuming that the technic of the fenestration procedure has been faultless, revision should not be undertaken (1) if no improvement in hearing occurs after operation; (2) if hearing has improved but not to practical levels in the speech frequencies, or (3) if the improvement of hearing recedes within four to twelve weeks. Should the last-mentioned event occur, the probable cause is fibrosis incited by inflammation or organization of blood having gravitated into the fenestra opening and perilymphatic spaces.

Revisions when indicated should be performed only after the cavity has become clean and well epithelized, thus obviating to a great extent the incidence of labyrinthitis. One may observe that after the improve-

14. Meltzer, P. E.: Revision of the Fenestration Operation, Arch. Otolaryng. **46**:528-533 (Oct.) 1947.

ment of hearing due to the fenestration operation depreciation may again develop without obvious cause as early as six to eight weeks postoperatively. Usually the interval is much longer. Presumably such an event is brought about by osteogenetic stimulation. In many instances suspicion is cast on a state of continuous aural discharge, which is conducive to a progressive inflammatory fibrosis and/or osteogenesis.

Successful revisions are relatively few in number, estimates and statistical appraisals accounting for about 15 to 25 per cent in prudently selected cases. In a small proportion of these improvement may be exhibited, but not to serviceable limits.

Considering the comparatively few successful results and the probable danger of irreversibly traumatizing the endolymphatic labyrinth in the process of recreating a fenestra, it would seem preferable to proceed according to the following sequence. For example, the hearing of the ear already operated on, the right, for instance, has disclosed audiometric evidence of fenestral closure. The auditory acuity of that ear has receded to preoperative levels which permit satisfactory adjustment to a hearing aid. Rather than consider revision of the right ear first, I would prefer to do the fenestration operation on the left ear, provided its hearing loss measures up to criteria which indicate favorable possibilities for serviceable improvement in hearing. Then, should the fenestra subsequently close and the resultant hearing loss not preclude the use of a hearing aid, revision of the right ear may be attempted with no additional risk to the patient's ultimate hearing capacity. On the one hand, this sequence of procedure offers the patient two possible opportunities, in addition to the original fenestration operation, to acquire hearing improvement; on the other, failure to revise the original fenestration operation without serious damage to the endolymphatic labyrinth would or should preclude further efforts to restore hearing in the opposite ear.

Furthermore, such a protocol of management offers the best and greatest possibilities of achieving effective improvement in hearing. With few exceptions, it is believed that sufficient cochlear response to a hearing aid must be assured in one ear before surgical restoration of hearing by revision is to be considered for the other.

CONCLUSION

Some of the aspects of clinical history, otoscopy and hearing tests which merit particular association with the process of selection of patients for fenestration surgery have been explored.

A method of selection based on pure tone audiometry has been contrived which is practical and relatively accurate, at least under the conditions which prevail with our own testing resources. With few qualifications this principle could be adapted for use in evaluating audi-

ometric results obtained in similar circumstances elsewhere. Obviously, however, varied technics and facilities would probably not permit close correlation among audiometric data extracted from different sources, but standards could be set up for individual systems.

A number of perplexities remain in connection with the clinical entity of otosclerosis and the problem concerning its histologic peculiarities. Despite the effort expended in both directions, the exact nature of its origin and development remains elusive. In the meantime, a great service may be done for those unfortunately afflicted with this condition. Perhaps it is fortunate for them that their loss of hearing should be of the type and limited magnitude for which there are effective palliative measures, alternatively the hearing aid or the fenestration operation or both.

To assure that the excellent performance of these measures will be maintained to the highest advantage, wisdom and intelligent guidance must direct the processes of selection of patients for fenestration surgery.

Mr. C. N. Hanley contributed to and assisted in the preparation of the statistical material.

CARCINOMA OF THE EAR

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CARCINOMA of the ear is not too frequently recorded in the literature. We believe, therefore, that a report should be made of 7 cases seen in the Department of Otology at Jefferson Hospital on the service of Dr. H. J. Williams in 1947 and 1948, during which time 1,883 new patients visited the clinic. In the same two years, 8 more cases were seen in other departments of the hospital. We shall also mention for the record, taken from a list of biopsies, a total of 65 carcinomas of the ear from all departments of the hospital from 1930 to 1948 inclusive.

According to Driver and Cole,[1] epithelioma of the auricle is frequently seen by the dermatologist and radiologist but seldom by the otolgist. These authors stated that "involvement of the auricle is common." In their series 5.5 per cent of all skin cancers during a period of twenty-five years involved the ear. However, they expressed the belief that primary lesions of the auditory canal are rare. The ninth annual report of the National Radium Commission,[2] for 1937 and 1938, lists basal cell carcinoma of the ear as representing 2.7 per cent of all skin cancers.

Newhart [3] in 1917 stated that "primary carcinoma of the middle ear was long regarded as one of the curiosities of medicine." As early as 1886, Kretschmann [4] collected 16 cases. In 1889, Zeroni [5] reported 5 cases of carcinoma of the ear, in 3 of which the external canal was

From the Department of Otology, Jefferson Medical College Hospital.
Read before the section on Otolaryngology of the College of Physicians of Philadelphia, April 27, 1949.

1. Driver, J. R., and Cole, H. N.: Treatment of Epithelioma of the Skin of the Ear, Am. J. Roentgenol. **48**:66, 1942.

2. Ninth Annual Report of the National Radium Commission, London, His Majesty's Stationery Office, 1937-1938.

3. Newhart, H.: Primary Carcinoma of the Middle Ear, Laryngoscope **27**:543, 1917.

4. Kretschmann, F.: Ueber Carzinome des Schläfenbeines, Arch. f. Ohrenh. **24**:231, 1886-1887.

5. Zeroni: Ueber das Carcinom des Gehörorgans, Arch. f. Ohrenh. **8**:141, 1899; cited by Furstenberg.[6]

involved and in 2 the tympanum. His very exhaustive study of the literature yielded 121 cases reported in the years 1804 through 1899. Levesque,[6] reporting a case in 1910, said that in the preceding literature he could find less than 50 authentic reports of carcinoma of the middle ear. Newhart,[3] in 1917, stated that since Zeroni's [5] monograph there were reports of only 34 cases of carcinoma of the middle ear. Broders,[7] in 1921, reported 63 cases; in approximately 84 per cent of these the auricle was involved, in 12 per cent the external auditory canal and in 1.5 per cent the middle ear. Furstenberg [8] in 1924 reported a case of primary adenocarcinoma of the middle ear and mastoid, with a discussion of the literature to that time. In 1935, Schall [9] reported on 15 neoplasms of the middle ear which were seen during a period of twelve years. Rosenwasser [10] in 1940 stated that the failure to report isolated cases may account for the scarcity of reports in the literature.

In 1941, Peele and Hauser [11] gave a very complete résumé of the subject of primary carcinoma of the external canal and middle ear. Warren and Gates [12] in 1941 described a carcinoma of ceruminous glands. Furstenberg,[8] Lukens,[13] Grabscheid,[14] Schall,[9] Fraser [15] and Thorell [16] have all reported cases of adenocarcinoma of the ear. In 1942, Figi and Hempstead [17] gave a very complete summary of the subject of malignant tumors of the middle ear and mastoid process,

6. Levesque: Des tumeurs malignes primitives de l'oreille moyenne, Gaz. méd. de Nantes **28**:89, 1910; cited by Newhart.[3]

7. Broders, A. C.: Epithelioma of the Ear: Study of Sixty-Three Cases, S. Clin. North America **1**:1401, 1921.

8. Furstenberg, A. C.: Primary Adenocarcinoma of the Middle Ear and Mastoid, Ann. Otol., Rhin. & Laryng. **33**:677, 1924.

9. Schall, L. A.: Neoplasms Involving the Middle Ear, Arch. Otolaryng. **22**:548 (Nov.) 1935.

10. Rosenwasser, H.: Neoplasms Involving the Middle Ear, Arch. Otolaryng. **32**:38 (July) 1940.

11. Peele, J. C., and Hauser, G. H.: Primary Carcinoma of the External Auditory Canal and Middle Ear: Review of Literature, Arch. Otolaryng. **34**:254 (Aug.) 1941.

12. Warren, S., and Gates, O.: Carcinoma of Ceruminous Glands, Am. J. Path. **17**:821, 1941.

13. Lukens, R. M.: Adenocarcinoma of the External Auditory Canal, Ann. Otol., Rhin. & Laryng. **45**:567, 1936.

14. Grabscheid, E.: Adenocarcinoma Involving the Middle Ear, Arch. Otolaryng. **37**:430 (March) 1943.

15. Fraser, J. S.: Malignant Disease of the External Acoustic Meatus and Middle Ear, Proc. Roy. Soc. Med. (Sect. Otol.) **23**:71, 1930.

16. Thorell, I.: Treatment of Malignant Tumors of the Middle Ear at Radiumhemmet, Stockholm, Acta radiol. **16**:242, 1935; abstracted, Brown, E. V. L.; Bothman, L., and Shambaugh, H. E.: Year Book of the Eye, Ear, Nose & Throat, Chicago, The Year Book Publishers, Inc., 1936, p. 358.

17. Figi, F. A., and Hempstead, B. E.: Malignant Tumors of the Middle Ear and Mastoid Process, Tr. Am. Acad. Ophth. (1942) **47**:210, 1943.

reporting 48 tumors from 1922 to 1941 and mentioning 500 others involving the pinna or external auditory canal. In 1946, Morrison [18] reported 13 cases of malignant tumors of the ear, including the external ear. Grossman, Donnelly and Snitman [19] in 1947 reported 6 cases of squamous cell carcinomas of the middle ear and mastoid. E. R. Garnett Passe [20] in 1948 reported a case of primary carcinoma of the eustachian tube and reviewed 2 other cases in the literature. Spencer [21] in 1935 wrote an outstanding article on this subject. Reports have been made by Robinson,[22] Buckman [23] and others.

Various authors have expressed their opinion as to the frequency of the occurrence of malignant tumors of the middle ear and mastoid process, their estimates ranging from 1 in 5,000 to 1 in 20,000 cases.

ETIOLOGY

A number of investigators have concluded that the following conditions may be precursors to carcinoma of the external ear: chronic eczema and other chronic dermatoses, trauma, frostbite, keratosis, sebaceous cyst, papilloma, chronic mastoiditis with polypi and chronic otorrhea, cutaneous horns, psoriasis, lupus vulgaris, xeroderma pigmentosa, burns and razor cuts. Figi and Hempstead [17] reported on 2 patients with carcinoma following repeated attacks of erysipelas with ulceration. Exposure to sunlight and ultraviolet rays, especially in persons of the white race, has been noted as a factor. Older age groups are more predisposed to the development of epithelioma, and white males more than white females. Newhart [5] and others have stated the belief that heredity plays the same role as it does in carcinomas of other parts of the body.

Carcinoma of the ear is very rare in Negroes. Quinland and Cuff [24] reported 300 cases of carcinoma in Negroes, but in not 1 case was the ear involved.

It is believed by some authorities that neoplasm is apt to develop on a background of degenerative changes.

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18. Morrison, L. E.: Cancer of the Middle Ear: Report of Four Cases, J. Indiana M. A. **39**:167, 1946.

19. Grossman, A. A.; Donnelly, W. A., and Snitman, M. F.: Carcinoma of the Middle Ear and Mastoid Process, Ann. Otol., Rhin. & Laryng. **56**:709, 1947.

20. Passe, E. R. G.: Primary Carcinoma of the Eustachian Tube, J. Laryng. & Otol. **62**:314, 1948.

21. Spencer, F. R.: Malignant Disease of the Ear, Arch. Otolaryng. **28**:916 (Dec.) 1938.

22. Robinson, G. A.: Malignant Tumors of the Ear, Laryngoscope **41**:467, 1931.

23. Buckman, L. T.: Carcinoma of the Middle Ear and Mastoid, Ann. Otol., Rhin. & Laryng. **52**:194, 1943.

24. Quinland, W. S., and Cuff, J. R.: Primary Carcinoma in the Negro: Anatomic Distribution of Three Hundred Cases, Arch. Path. **30**:393 (July) 1940.

According to Peele and Hauser,[11] the etiology of carcinoma of the middle ear and mastoid has not been definitely settled by scientific facts. But Newhart,[3] Barnes [25] and others have expressed the belief that epithelial changes take place in the middle ear because of trauma and chemical irritation from a chronic discharge and also because of invasion of epithelium from the auditory canal into the middle ear. Therefore chronic otorrhea and aural polypi should always keep one alert to the possibilities of carcinoma.

SYMPTOMATOLOGY

Symptoms and signs of carcinoma of the external ear consist, for the most part, of a tumor or ulcerated area with bleeding that may enlarge slowly or rapidly, eventually invading surrounding tissues. Severe pain follows later, although many believe that pain may be an early feature and far out of proportion to the amount of pathologic change evident in the ear.

Symptoms of carcinoma of the middle ear and mastoid may generally be enumerated as the following, according to most writers:

1. "A complaint of persistent, deep-seated intractable pain about the ear, severe otalgia unexplained by any visible pathologic changes in the tympanum, the posterior group of paranasal sinuses, the nasopharynx, the teeth or the larynx," according to Stokes.[26] In later stages there may be excruciating pain on chewing. The pain radiates along the course of the trigeminal nerve.

2. The presence of a bloody discharge, having a tendency to be obstinate and regular.

3. Ulceration of the canal of unexplained origin.

4. Aural growths or polypi or tough resistant granulations which recur rapidly when removed and tend to bleed on manipulation.

5. "The occurrence of a supposedly commonplace lesion of the external or middle ear, not only becoming refractory to treatment but showing in its inexorable advance a baffling dissimilarity to the usual clinical course," according to Stokes.[26]

6. Palsy of the various cranial nerves, most commonly of the facial and at times recurrent in type or the jugular foramen syndrome—that is palsy of the ninth, tenth, eleven and twelfth nerves and then the abducens and oculomotor.

7. Deafness, vertigo, mastoid tenderness and tinnitus, occurring less frequently than the previously mentioned symptoms.

DIAGNOSIS

In making a diagnosis of carcinoma of the ear, one must bear in mind the history, the symptoms and signs and the clinical findings. Biopsy is essential, and it should be repeated if the findings are not positive for carcinoma. Roentgenograms should be made of the temporal

25. Barnes, E. B.: Carcinoma of the Ear, Proc. Roy. Soc. Med. (Sect. Otol.) **23**:1231, 1930.

26. Stokes, H. B.: Primary Tumors of the Temporal Bone: Report of a Case, Arch. Otolaryng. **32**:1023 (Dec.) 1940.

Fig. 1.—Squamous cell carcinoma of intermediate grade, showing large areas of squamous cells with moderate amounts of eosinophilic cytoplasm and typical pearl formations. There are a number of mitotic figures. Between the groups of squamous cells can be seen areas of inflammatory reaction. × 100.

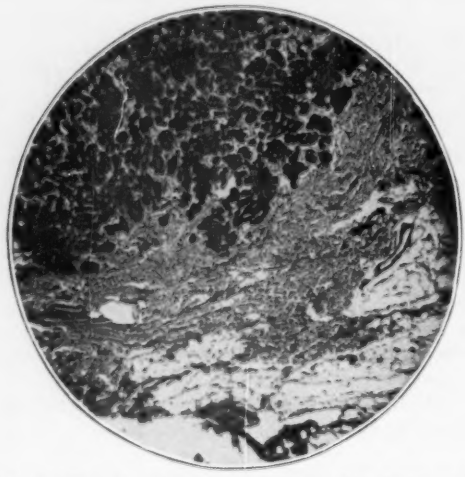

Fig. 2.—Basal cell carcinoma. The upper left portion shows an area densely infiltrated with nests of basal cell carcinoma, forming abundant islands completely replacing the former tissue. Below there are smooth muscle strands with areolar tissue and a few inflammatory cells between the tumor and adjacent tissue. × 50.

bone and other bones of the skull and also of other areas of the body to determine the presence of possible metastasis in late cases. Serologic examinations and cultures are indicated. Surgical exploration of the mastoid process and middle ear should be done in all cases of suspected malignancy.

The histologic types of tumors of the external canal and middle ear are listed in the accompanying table.

Histologic Types of Tumors of the External Auditory Canal and Middle Ear

1. Squamous cell carcinoma	5. Endothelioma	11. Neurofibroma
2. Basal cell carcinoma	6. Hemangioendothelioma	12. Adenocarcinoma
3. Basosquamous cell carcinoma	7. Lymphoendothelioma	13. Carcinoma of ceruminous glands
4. Epithelioma adenoides cysticum	8. Sarcoma	14. Dermoid
	9. Fibrosarcoma	15. Parotid cell nest
	10. Myxosarcoma	

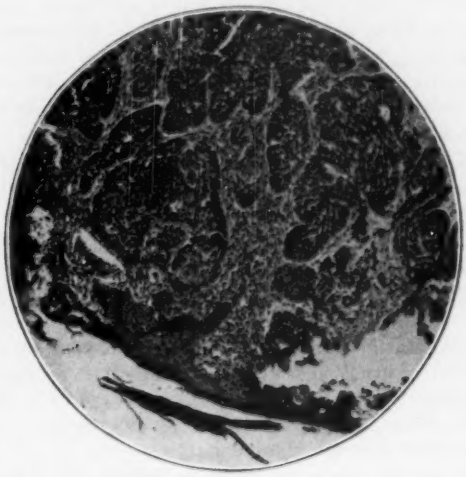

Fig. 3.—Basosquamous cell carcinoma. In the lower left portion there is normal epithelium of squamous cells with keratinization. From this area can be seen finger-like projections of cells and dark-staining nuclei, the beginning of early squamous cell carcinoma. In the dermis are large irregular nests of dark-staining cells which resemble the basal cell layer of the epidermis. In the islands of cells is keratinized material. Hyperchromatic nuclei with epithelial pearls are present. There can also be seen an inflammatory reaction with plasma cells and lymphocytes which accompany carcinoma. × 100.

TREATMENT

Surgical.—Surgery has been an effective method of treatment throughout the years if the lesion is seen early, and will continue to be so, but it must be radical enough to remove all the tumor, which at times is mutilating and disfiguring to the individual.

In 1947, Mohs [27] reported treatment of 107 patients with carcinoma of the ear in a nine and one-fourth year period by a chemosurgical method. The results depended on the size of the lesion. He reported 91.4 per cent cures in a group of patients who had not been given previous treatment. The chemosurgical technic, by which cancer of the ear may be excised under systematic microscopic control, has proved effective but entails the use of a well trained staff.

Schall [9] stated that conservative surgical procedures have no place in the treatment of aural neoplasms.

Stokes,[28] in 1940, got the impression from the literature "that the management of malignant growths of the temporal bone is distressingly inadequate; that the prognosis is very grave, since early diagnosis is exceptional, and that the mortality remains almost unchanged over the years in spite of all modern facilities for diagnosis and treatment."

Morrison [16] in 1946 stated that "the risk of intracranial complications is greatly increased by the conservative approach" to treatment.

Figi and Hempstead [17] (1943) concluded that if carcinoma of the middle ear and mastoid is recognized reasonably early and is dealt with adequately the prognosis is as satisfactory as that generally offered by malignant tumors of the nasal sinuses.

Irradiation.—Driver and Cole,[1] in a follow-up study of 107 of 130 treated patients with skin cancer of the ear, found 87.9 per cent cures from one to five years.

Pfahler and Vastine [28] expressed the opinion that a greater percentage of failures occur in the treatment of patients with cancers in the region of the ear than in any other relatively superficial portion of the body.

Martin and Martin [29] stated "no single measure has been universally successful in our hands and various combinations of electrosurgery, roentgen therapy and radium therapy have been used." They said: "Poor results following surgery alone are due to extension of the malignant process into the tissues adjacent to the ear. . . ." They stated the belief that a plan of irradiation should be instituted to follow surgery and that "extension of the malignant process into the underlying bone makes the prognosis very poor and no cases of this type have been completely cured in our clinic."

Treatment of the Middle Ear and Mastoid.—Schall [9] stated that any surgical procedure less radical than the removal of the entire cutaneous

27. Mohs, F. E.: Chemosurgical Treatment of Cancer of the Ear: A Microscopically Controlled Method of Excision. Surgery **21**:605, 1947.

28. Pfahler, G. E., and Vastine, J. H.: The Treatment of Cancer in the Region of the Ear, Am. J. Roentgenol. **37**:350, 1937.

29. Martin, C. L., and Martin, J. A.: Treatment of Advanced Cancer Involving the Ear, Am. J. Roentgenol. **60**:750, 1948.

canal is inadequate. "Radical mastoidectomy . . . should be performed for inspection of the middle ear and the cavity of the mastoid and to secure sufficient space for the proper application of radium."

In involvement of the mastoid, it is evident from our poor results that as much as possible of the temporal bone should be removed.

SUMMARY OF CASE MATERIAL

Of our 7 patients, 6 had squamous cell carcinoma and 1 a basosquamous cell carcinoma. Ages were 32, 39, 46, 53 and 72 years (3 patients). All were of the white race. One was a pottery worker; 2 were housewives; 1 was a farmer; 1, a sheet metal worker; 1, a plumber, and 1, a brick pointer and plasterer. Five were males, and 2 were females. Five had chronic suppurative otitis media, and 2 gave a history of trauma. One patient had had an epithelioma removed from the eyelid, and 1 reported cancer in the family.

Original sites of the disease, as far as we could estimate, were as follows: the auricle, 1 case; the auditory canal, 2 cases; the auricle and canal, 1 case, and the middle ear or mastoid, 3 cases.

Symptoms.—By the time we saw them, 5 patients already showed invasion by the disease process of other structures of the ear or temporal bone. Six had pain; 5, discharge; 2, ulceration; 5, deafness; 2, bleeding; 3, facial paralysis; 1, recurring facial paralysis; 1, vertigo, and 3, mastoid tenderness. None had tinnitus; 2 had swelling in the canal or about the ear; 2 had hoarseness and/or dysphagia, and 1 had loss of weight.

Clinical Findings.—Four patients had paralysis of the cranial nerves; 2, involvement of tissues about the ear; 6, involvement of the external auditory canal; 5, discharge; 5, tumor in the canal; 4, ulceration; 4, fixation of the lesion over the mastoid process or to the canal wall; 3, facial paralysis; 1, perforation of the auricle; 3, mastoid tenderness, and 3, palpable nodes.

Roentgen Findings.—The roentgenogram of 1 patient showed no evidence of pathologic change, and that of 1, evidence of previous radical mastoid operation only. Secondary cancerous invasion of facial and skull bones was shown on the roentgenogram of another and sclerosis of the mastoid was shown on the roentgenograms of 2. Roentgenograms were not made for 2 patients, 1 of whom had had a previous operation.

Previous Treatment.—Three patients had had radical mastoidectomy; 1, a radical mastoidectomy with removal of a tumor of the canal and roentgen therapy; one, conservative local and medical treatment; one, several surgical excisions plus therapy, and 1, no treatment.

Our Treatment.—Wide excision of the auricle and the auditory canal was done on 1 patient, and section of cranial and cervical nerves for pain on 1; radical mastoidectomy and removal of the canal and auricle was performed on another; 1 was treated by radical mastoidectomy and removal of the canal plus roentgen therapy, and a secondary mastoidectomy and section of cranial nerves and ligation of the carotid artery was performed on 1. Two patients were given no treatment because the disease was too far advanced.

Fig. 4 (case 1).—Areas of swelling, with ulceration and draining fistulas (indicated by the arrows).

REPORT OF CASES

CASE 1.—*Squamous cell carcinoma.*

J. B., a 32 year old white man entered Jefferson Hospital on Sept. 26, 1948 because of hoarseness and dysphagia, emaciation, pain and swelling over the right ear, face and temporal region, two draining fistulas and foul otorrhea, following a radical mastoidectomy in May 1948 for a chronic suppurative otitis media existing since childhood.

Examination revealed paralysis of the right vocal cord and paralysis of the ninth, tenth, eleventh and twelfth cranial nerves. Roentgenograms showed destructive lesions of the mastoid process, petrous pyramid, squamosa, occipital bone, zygoma and maxillary bone.

Fig. 5 (case 1).—*A*, large area of destruction of the right side involving the mastoid area, the petrous pyramid, the squamous portion of the occipital bone and part of the zygoma; *B*, view from a slightly different angle, showing involvement of the mastoid area and zygoma.

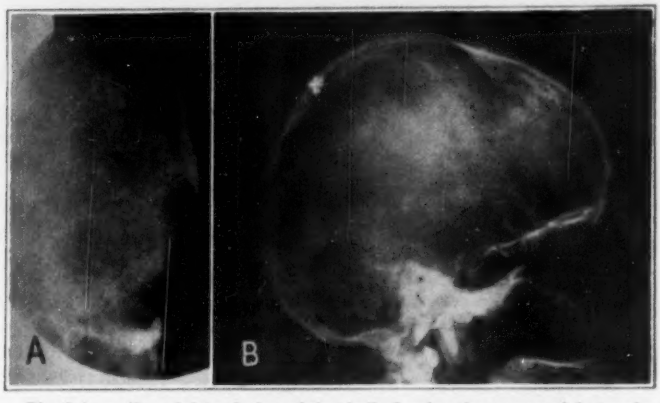

Fig. 6 (case 1).—*A*, lateral view of the skull, showing the amount of destruction of bony structures. *B*, a different view, showing the destruction of the cortex of the mastoid area.

The origin of this lesion was probably in the middle ear and mastoid process. The disease was too far advanced for medical aid, and the patient died on November 17.

CASE 2.—*Squamous cell carcinoma.*

C. H., a white man aged 39 years, was admitted to Jefferson Hospital on Sept. 24, 1947 because of pain in the right ear, present since before a radical mastoidectomy in September 1946, and continuous otorrhea, present since childhood. A revision of the mastoidectomy on April 30, 1947 and laboratory examination had revealed carcinoma, and 2,600 r of roentgen radiation was given.

Cervical adenopathy developed, and because of intractable pain Dr. Jaeger severed the fifth and ninth cranial nerves and the nervus intermedius on September

Fig. 7 (case 4).—*A*, paralysis and muscular atrophy of the right side of the face of a patient with carcinoma of the ear; *B*, large cancerous mass filling the auditory meatus and spreading to the concha.

29 and ligated the external carotid artery on October 13. The patient died about May 1948.

CASE 3.—*Basosquamous cell carcinoma.*

T. P., aged 72 years, a white man, was admitted to Jefferson Hospital April 10, 1948 because of constant intolerable pain in and about the left ear, which had followed roentgen irradiation five weeks before, and foul otorrhea.

In 1921 he sustained ammonia burns of the left auricle. In May 1936 roentgen irradiation was given because the wound had failed to heal. In November 1936 the lobe, tragus and three quarters of the auditory canal were removed because of squamous cell carcinoma, and plastic repair was done. In October 1945 a recurrent lesion was excised. In January 1946 the rest of the auricle was excised because of recurrence of the carcinoma. The patient was well for over two years. There was weakness of the left side of the face and deviation of the tongue to the left.

On April 14, 1948 the fifth, eighth and ninth nerves and the nervus intermedius were sectioned, as were also the roots of the second, third and fourth cervical nerves because of pain. The patient died soon after his return from the operating room.

CASE 4.—*Squamous cell carcinoma.*

M. M., a 72 year old white woman, was admitted to Jefferson Hospital on Sept. 20, 1948 with pain and hemorrhage from the right ear and a purplish red, tender cobblestone mass 2.5 cm. in diameter filling the canal and concha. Also present

Fig. 8 (case 5).—Results of use of plastic flaps to cover the area exposed by removal of the auricle and adjacent tissue.

was a complete right peripheral facial paralysis with muscle atrophy, and dysphagia of three and one-half years' duration. The mass, which had grown rapidly during the preceding two months, had started as a very small nodule in 1945. Radical mastoidectomy had been done twenty-one years before this admission for long-standing otorrhea. There were three attacks of facial paralysis, two of which had cleared up. Because of the advanced disease and a poor risk treatment was not instituted.

CASE 5.—*Squamous cell carcinoma.*

L. G., aged 53, a white man, was admitted to Jefferson Hospital on Oct. 1, 1947 with a large ulcerating lesion of the lower fourth of the auricle, extending through

to the posterior surface and involving the auditory canal. The origin was a traumatized "blackhead" one and one-half years previously.

Very wide surgical excision of the auricle was done, together with excision of the auditory canal. Skin grafting was done later. The last follow-up examination, on May 18, 1949, showed no recurrence, and the patient was in good health.

CASE 6.—*Squamous cell carcinoma.*

B. L., aged 72 years, a white woman, was admitted to Jefferson Hospital Feb. 26, 1948 with an inoperable metastatic egg-sized mass below the right ear, of four months' duration. There was a clean, entirely healed radical mastoidectomy cavity and facial paralysis. On March 3, 1947 a radical mastoidectomy had been performed, with removal of the entire auditory canal, because of a cancerous growth in the canal. Partial facial paralysis existed before operation, and paralysis was complete after operation. The whole facial ridge was eroded and replaced with granulations. An incomplete course of roentgen therapy followed this operation. For sixteen months before operation there was otorrhea and headache and mastoid tenderness. There was a history of otitis media and mastoiditis without operation thirty years previously.

An incompleted course of roentgen therapy was given to the mass in the neck. The patient died at home on June 6 after a cerebral hemorrhage.

CASE 7.—*Squamous cell carcinoma.*

W. G., a white man aged 46, was admitted to Jefferson Hospital Dec. 3, 1948 with pain in the left ear, foul otorrhea, bleeding, crusting, mastoid tenderness and vertigo, present for three months, and a cancerous tumor in the auditory canal. Chronic otorrhea had existed for forty years. In 1926 an aural polyp had been removed. On Dec. 6, 1948 the auditory canal was completely excised and a radical mastoidectomy was done, revealing erosion of the proximal end of the facial ridge. Facial paralysis gradually developed. Because of severe pain, a wide excision of the auricle was done on Feb. 14, 1949, and the mastoidectomy was revised, revealing total disintegration of the distal half of the facial ridge, the entire tip, the bony floor of the canal and the floor of the middle ear. The anterior wall of the bony canal was removed as a sequestrum. All these parts, plus tissue from the eustachian tube, showed squamous cell carcinoma. Roentgen radiation (2,800 r) was given to relieve pain. From April 25 to May 19 he received 3,000 r of roentgen rays for extension to cervical nodes. This receded.

On July 29 a prefrontal lobotomy was done for pain. On September 1, he was still having pain, and word of his death was received later.

SUMMARY OF CASES FROM ALL DEPARTMENTS OF JEFFERSON HOSPITAL

Following is a brief summary of 65 cases of carcinoma of the ear from all departments of Jefferson Hospital from 1930 through 1948. The pathologic diagnosis (from biopsy specimens) was squamous cell carcinoma, not graded, 27 cases; squamous cell, low grade, 10 cases; squamous cell, intermediate grade, 7 cases; basal cell, 16 cases, and basosquamous cell, 4 cases. The type of neoplasm was undetermined in 1 case. Of the 65 cases there were histories available in only 43, from which the following information is given. In 2 cases, the patient's

age was from 30 to 40 years; in 3 cases, from 40 to 50; in 11 cases, from 50 to 60; in 9 cases, from 60 to 70; in 13 cases from 70 to 80, and in 5 cases, from 80 to 86. The patients comprised 33 men and 10 women, of whom 42 were white and 1 was Negro.

The symptoms and signs in these 43 cases were distributed in the following manner: otorrhea, 13; hemorrhage, 11; pain, 11; ulceration, 19; deafness, 5; vertigo, 1, and mastoid tenderness, 1. They were present in 3 patients from one to six months; in 4, from six months to one year; in 10, from one to two years; in 6, from two to three years; in 3, three years; in 4, four years; in 2, five years; in 1, six years; in 4, ten years; in 5, from ten to twenty-five years, and in 1, for an unknown length of time.

Involvement of the seventh cranial nerve was noted in 3 cases; of the seventh, eighth, ninth, tenth, eleventh and twelfth cranial nerves, in 2; of the first, second and third cervical nerves and the fifth and ninth cranial nerves, in 1, and of the ninth, tenth, eleventh and twelfth cranial nerves, in 1.

The lesions were located in the auricle in 27 cases; in the auricle and canal in 2; in the auricle and temporal bone in 2; in the canal in 6; in the mastoid process in 2, and in the canal and mastoid process in 4.

There are no follow-up records for most of the 65 cases reported. Thirteen patients are known to have died, but we have no record of the causes of death. One died within one month; 1, within two months; 1, within a few months; 1, within six months; 2, within one year; 1, within one and one-half years; 1, within two years; 1, within three years; 2, within four years; 1, within five years, and 1 within seven years. Nine patients died of known causes. Two died of cerebral thrombosis and cerebral hemorrhage after seven and four months, respectively; 1, of postoperative anesthesia; 1, of pulmonary metastasis within one year; 1, of pulmonary edema within three months; 1, of general debility within four months; 1, of coronary occlusion within four and one-half years; 1, of heart disease within eight and one-half years, and 1, in a railroad accident within four years.

SUMMARY AND CONCLUSIONS

The literature on carcinoma of the ear is partially reviewed for views and opinions on occurrence, etiology, symptoms and treatment of the disease.

Seven cases of carcinoma of the ear observed in the ear clinic of Jefferson Hospital during the two years 1947 and 1948 are reported, and reports of 65 biopsies of carcinoma of the ear from all departments of the hospital since 1930 are summarized.

We are impressed by the fact that there is too frequently delay in diagnosis on the part of the physician and too often the patient delays before consulting a physician.

Treatment has been too conservative, both on our part and on that of others.

All physicians should be more alert to discover very early signs and symptoms of carcinoma of the ear. They should especially observe closely ears with chronic discharges.

The pathology and x-ray departments and the tumor clinic of Jefferson Hospital cooperated in making available material and data for this study.

SITE OF LESION IN PARALYSIS OF TWELFTH AND/OR SEVENTH CRANIAL NERVE

KURT TSCHIASSNY, M.D.
CINCINNATI

INTERESTING observations in a case of bilateral complete paralysis of the tongue (panglossoplegia) were the occasion for these studies. New information resulting therefrom is presented concerning particular aspects of the neural and muscular mechanisms responsible for certain movements of the tongue and of the lips. On the basis of these findings in correlation with known anatomic factors differential topognosis of the lesion in instances of hypoglossal and/or facial nerve paralysis is discussed.

REPORT OF A CASE

A 57 year old white housewife was referred to the Cincinnati General Hospital by her family physician on Feb. 2, 1947 because of inability to chew, to swallow and, particularly, to talk in an understandable manner. It was the opinion of her physician that all her troubles were caused by a complete bilateral paralysis of the tongue. The illness had started in November 1945, with difficulty in pronouncing all the linguals and certain of the labials, particularly "p" and "b". In June 1946, the difficulties in chewing and swallowing started. They appeared to be caused directly by inability to perform proper tongue movements. After November 1946, the muscles of the lips became weak; the corners drooped, and saliva drooled down from the mouth. The patient occasionally noticed a muscular tic around the mouth. Slight disturbance in tasting food was felt. She lost almost 30 pounds (13.6 Kg.) in weight. Otherwise no signs or symptoms were noticed.

On physical examination a pronounced dysarthria was observed which was in conformity with the speech trouble as described. The tongue was atrophic and wrinkled; it lay flat on the floor of the mouth, and there was fasciculation all over the tongue. The patient was unable to produce even a slight motion of the tongue in lateral or vertical planes. When she was asked to protrude the tongue, it moved forward in the midline only as far as the inner margin of the lower teeth. The total movement of the tongue was roughly 0.5 cm. (fig. 1). The corners of the mouth drooped bilaterally, probably more noticeably at the right. The musculature of the lips appeared to be atrophic, and there was a marked tremor around the lips involving apparently the orbicularis oris muscle. Muscles of mastication and the palate, pharynx and larynx appeared normal. Taste and common sensation were normal. There was fasciculation of both upper and the right lower forearms and atrophy of the thenar and hypothenar eminences. Gag reflexes and

This paper was read before the Cincinnati Otolaryngological Society on May 2, 1949.

From the Departments of Neurology and Otolaryngology, Cincinnati General Hospital and the University of Cincinnati College of Medicine.

deep reflexes were hyperactive. Cochlear and vestibular function was normal. Laboratory data were not contributory. The diagnosis offered by the neurologic service was progressive bulbar paralysis, probably related to amyotrophic lateral sclerosis. The condition of the facial musculature was regarded as indicative of a slight weakness of the facial nerve of the upper motor neuron type. The patient was discharged to the care of her private physician on the fifth hospital day.

Two features of this case appeared to present unanswered problems: (1) the character of the facial palsy, which appeared to be restricted to paralysis of the orbicularis oris muscle, and (2) the presence of some voluntary movement of the tongue in spite of complete paralysis of the twelfth nerve bilaterally.

Fig. 1.—The attempt to protrude the tongue as far as possible succeeds in bringing it only as far as the teeth. This movement represents the range of action of the geniohyoid muscles.

THE FIRST PROBLEM

The interpretation of the paralysis of the orbicularis oris muscle as a manifestation of a facial nerve lesion of the central type seemed questionable, since atrophy and fasciculation are not assumed to be associated with an upper motor neuron lesion. In addition, such a selective lesion of a single muscle seemed hard to conceive of in terms of involvement of the pyramidal tract. An answer to the problem may be found in the "Compendium of Regional Diagnosis in Lesions of the Brain and Spinal Cord" by Bing.[1] These authors stated: "A lesion of the hypoglossal

1. Bing, R.: Compendium of Regional Diagnosis in Lesions of the Brain and Spinal Cord: A Concise Introduction to the Principles of Localization of Diseases and Injuries of the Nervous System, ed. 11, translated and edited by W. Haymaker, St. Louis, C. V. Mosby Company, 1940, p. 152.

nucleus produces not only a lingual paralysis but also a homolateral orbicularis oris paralysis. Infranuclear hypoglossal lesions on the other hand do not affect the orbicularis oris muscle. We have to assume, therefore, that the hypoglossal nucleus participates with the facial nucleus in supplying the orbicularis oris. The pathway of fibres passing from the hypoglossal nucleus to the facial nerve is not yet anatomically proven" (fig. 2).

This statement affords a somewhat modified corroboration of the comment in Piersol's Anatomy [2] that by "clinical and experimental evidence a connection is probably established by the posterior longitudinal bundle between the nucleus of the hypoglossal nerve and that of the

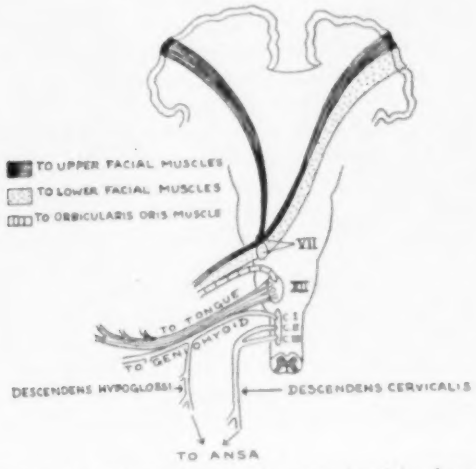

Fig. 2.—Note the bilateral—crossed and uncrossed—origin of pyramidal tract fibers responsible for innervation of upper facial muscles and the unilateral—crossed only—origin for innervation of the lower facial muscles. Fibers responsible for the nerve supply of the orbicularis oris muscle originate in the hypoglossal nucleus and innervate the geniohyoid muscle. They also are responsible for the descendens hypoglossi. The twelfth nerve is joined by fibers which originate in the first cervical nucleus. This nerve forms the ansa hypoglossi by anastomosis with the descendens cervicalis, which originates in the second and third cervical segment. (Enlarged and modified from Bing.[1])

facial nerves whereby the closely associated movements of the lips and tongue are assured."

In the present case the bilateral facial paralysis restricted to the orbicularis oris muscle, evidently of lower motor neuron type, appears satisfactorily explained on the basis of the anatomic conditions men-

2. Piersol, G. A.: Human Anatomy, Philadelphia, J. P. Lippincott Company, 1907, p. 1117.

tioned by these authors. It should be emphasized that orbicularis oris paralysis must be differentiated from the "lower face weakness," which it may sometimes resemble. While it is our custom to regard the latter as a manifestation of an upper motor neuron lesion of the seventh cranial nerve, the former, particularly when present selectively and associated with glossoplegia, should be regarded as indicative of a lower motor neuron lesion of the twelfth nerve.

Testing the Orbicularis Oris Muscle.—The action of this muscle is manifested particularly by the ability to pucker and pronounce explosive labials, such as "b" and "p." In bilateral paralysis of the orbicularis oris, drooling of saliva and pronunciation of the fricative labials "f" and "v" instead of "p" and "b" is significant. The latter phenomenon can be demonstrated by a simple experiment. On placing the finger in either corner of the mouth an attempt to pronounce the words "bobby" and "pepper" results in pronouncing "vovvy" and "feffer." In this maneuver, by which conditions analogous to orbicularis oris paralysis are effected, the explosive labials are replaced by the fricatives.

Application for Regional Diagnosis.—A. In Hypoglossal Paralysis: The orbicularis oris muscle occupies an exceptional position among the muscles supplied by the facial nerve. The nerve fibers responsible for stimulation of this muscle are the only ones in the course of the motor seventh nerve which do not (or mainly not) originate in the nucleus of the seventh but in the nucleus of the twelfth nerve. On the basis of this anatomic peculiarity some consideration concerning topognosis of the nerve lesions are presented. In hypoglossal paralysis Bing [3] mentioned the condition of the orbicularis oris muscle as a factor useful for differentiation between a nuclear and an infranuclear lesion of the twelfth nerve, this muscle being paralyzed in the former but not being involved in the latter.

B. In Facial Paralysis: In my opinion, the condition of this muscle may, on the same basis, offer a factor for differential topognosis in instances of facial nerve paralysis. Since the majority of nerve fibers responsible for the orbicularis oris branch of the seventh nerve do not originate in the nucleus of the seventh but join this nerve only below its nucleus, it can be assumed that the action of this muscle is not interfered with in upper motor neuron and/or in nuclear lesions of the seventh nerve but is necessarily involved in infranuclear lesions.

Characteristics Significant for the Upper Motor Neuron Type of Facial Paralyses.—The syndrome of the upper motor neuron lesion of the seventh nerve includes the following characteristics: 1. Unimpaired action of the upper facial muscles including the orbicularis palpebrarum

3. Bing, R.: Textbook of Nervous Diseases, edited and enlarged by W. Haymaker, St. Louis, C. V. Mosby Company, 1939, pp. 97-98.

muscles due to their bilateral cortical representation (fig. 2), the former being manifested by symmetric winking and the latter by the absence of Bell's phenomenon. 2. Unimpaired action of the muscles of expression when stimulated by emotion, since the impulses to the muscles do not then pass over the pyramidal tracts. Figure 3 offers an instructive illustration of this phenomenon; the patient had a vascular lesion in the third frontal convolution at the left. 3. Unimpaired blinking and stapedius reflexes. The latter is manifested by absence of phonophobia (hyperacousis) on loud acoustic stimuli. 4. Unimpaired taste sensation on the anterior two thirds of the tongue. 5. Unimpaired lacrimation

Fig. 3.—*A*, taken when the patient was told: "Close your eyes and show me your teeth." Note the approximately equal distribution of the wrinkles over the forehead, the absence of Bell's phenomenon and definite paralysis of muscles of lower part of face. *B*, taken after saying: "Let's have another picture for your sweetheart." Note the absence of paralysis of muscles of expression on emotion, since this time the stimulus passed over extrapyramidal tracts.

and salivation. 6. Unimpaired action of the orbicularis oris muscle. This can be considered as an additional sign.

Condition of the Orbicularis Oris Muscle in Topognosis of Infranuclear Facial Paralysis.—In a lower motor neuron lesion of the facial nerve, examination of the orbicularis oris muscle may be particularly helpful in differentiating a lesion located in the nucleus of the seventh nerve from one located below the branching of the chorda tympani (infrachordal). In a case of a facial nerve lesion in either one of these locations taste sense and lacrimation are preserved. So far differentiation has been dependent exclusively on testing for the presence or

absence of the stapedius reflex, its absence (manifested by phonophobia) being indicative of a nuclear lesion and its presence indicative of a lesion below the branching of the stapedius nerve, occurring of course, when taste sensations are preserved below the chorda tympani.[4] The presense or absence of the orbicularis oris action may represent an additional criterion for differentiation between lesions in these two locations. In instances of peripheral facial nerve paralysis with unimpaired taste and lacrimation the presence of the orbicularis oris action indicates a nuclear lesion; contrariwise, the absence, an infrachordal location.

In the table is presented a schematic summary including seven different syndromes characteristic of facial nerve lesions at seven different levels in the course of the nerve. This represents an enlargement of a similar scheme presented in a previous article,[5] in which, however, only four levels were differentiated.

Signs Characteristic of Facial Nerve Lesions at Various Levels

Level	Upper Face Movement	Bell's Phenomenon	Emotional Movement	Blinking Reflex	Stapedius Reflex	Orbicularis Oris Movement	Tears	Taste
Supranuclear	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes
Nuclear	No	Yes	No	No	No	Yes	Yes	Yes
Suprageniculate	No	Yes	No	No	No	No	No	Yes
Transgeniculate	No	Yes	No	No	No	No	No	No
Suprastapedial	No	Yes	No	No	No	No	Yes	No
Infrastapedial	No	Yes	No	No	Yes	No	Yes	No
Infrachordal	No	Yes	No	No	Yes	No	Yes	Yes

THE SECOND PROBLEM

The second problem is centered around the question of why in a case of bilateral complete paralysis of the twelfth nerve some restricted mobility of the tongue remained. The assumption that both hypoglossal nerves were completely paralyzed in the present case was based on the following facts: 1. There was not even slight motion when the patient was asked to move the tongue in any direction except forward. 2. The atrophy of the tongue, its wrinkled appearance, and the fasciculation were indicative of an advanced lower motor neuron lesion. Unfortunately, the electric reaction of degeneration was not tested for. The patient was presented at the neurologic conference on Feb. 8, 1947 and discussed with Drs. Harry M. Salzer, I. Mark Scheinker and me. The entry in the chart reads as follows: "Dr. Salzer's impression:

4. Tschiassny, K.: Herpes Zoster Oticus (Ramsay Hunt's Syndrome): Comments on an Article by Johnson and Zonderman, Arch. Otolaryng. **51**:73 (Jan.) 1950.

5. Tschiassny, K.: The Site of the Facial Nerve Lesion in Cases of Ramsay Hunt's Syndrome, Ann. Otol., Rhin. & Laryng. **55**:152 (March) 1946.

Progressive bulbar palsy probably related to amyotrophic lateral sclerosis. Dr. Scheinker believes the entire picture is compatible with the diagnosis amyotrophic lateral sclerosis. Dr. Tschiassny pointed out that the patient's ability to move the tongue is not due to function of the tongue muscles but to action of the geniohyoid muscles, which are innervated by cervical nerves."

My statement at this conference was based on the presence of a slight forward movement of the hyoid bone which was felt to occur when the patient was asked to protrude the tongue as far as possible. It was, however, left for future studies to support my impression by objectively demonstrable findings. No applicable information concerning this was obtained by consulting current textbooks. Most of the authors assert that in complete bilateral paralysis of the twelfth nerve the tongue cannot be protruded at all but lies flat on the floor of the mouth. Only Kinnier Wilson,[6] in his textbook of neurology, mentioned that in double glossoplegia "the tongue cannot be put out beyond the teeth." This remark by implication conforms to some extent with observations in the present case. It was left for special studies on the neural and muscular mechanisms responsible for possible lingual movements to prove the explanation which was offered at the clinical conference mentioned above.

Anatomic Review.—A brief review of the anatomy of the structures responsible for movements of the tongue may precede the description of those studies. The seven pairs of tongue muscles are classified as comprising two groups, the four intrinsic and the three extrinsic muscles. The former, designated according to the plane of extension of their fibers, are mainly responsible for shaping the tongue. The latter, designated according to the bones of their origin, are responsible for the gross movements of the tongue. The genioglossus muscle pulls the tongue out of the mouth and to the opposite side; the styloglossus pulls it back and up toward its own side; and the hyoglossus pulls it back and down toward its own side. The glossopalatine and glossopharyngeal muscles (lowermost portion of the superior pharyngeal constrictor) are not muscles of the tongue but are muscles of the palate and of the pharynx respectively.

The lower surface of the base of the tongue is firmly attached to the hyoid bone by a layer of strengthened connective tissue, sometimes referred to as the hyoglossal membrane. By this firm attachment any movement of the hyoid bone activated by muscles attached to it will necessarily cause a passive movement of the tongue in an identical plane and direction, and, vice versa, any movement of the tongue activated

6. Wilson, S. A. K.: Neurology, edited by A. N. Bruce, Baltimore, Williams & Wilkins Company, 1940, vol. 1, p. 425.

by the extrinsic lingual musculature will cause a passive movement of the hyoid bone and of the structures suspended on this bone. This statement conforms with daily experience in performing indirect laryngoscopy. Pulling the tongue out of the mouth raises the larynx. Among the muscles attached to the hyoid bone there are three in particular which are responsible for its upward and forward movement: the digastrics, the mylohyoid and the geniohyoids. The digastrics and the mylohyoid, however, are mainly elevators, their action in pulling the hyoid bone forward being of minor importance. This becomes evident from the site of their attachment at the mandible and the plane of their extension. Contrariwise, the geniohyoid, extending in an almost anteroposterior direction, is more effective in pulling the hyoid bone forward and only to a minor degree in pulling it upward. In all these actions the mandible represents the point of fixation. Of course, these muscles also act in the opposite direction, pulling the mandible down when the hyoid bone is kept fixed.

The motor nerve supply of the tongue muscles is provided exclusively by the hypoglossal nerve, its neurons originating in the medullary nucleus. The nerve, just beyond its exit from the skull, is joined by another nerve structure which originates from the first and probably the second cervical nerve, branching off from the loop connecting the two of them.[7] These fibers, representing a sort of spinal portion of the hypoglossal nerve in its extracranial course, are in no way concerned with the muscles of the tongue. They are responsible for the nerve supply of the geniohyoid and thyrohyoid muscles only. Branching from the hypoglossal nerve at the site where it crosses the occipital and/or the internal carotid artery are nerve fibers passing downward, known as the descendens hypoglossi. Likewise, from the second and third cervical motor segment a bundle of nerve fibers passes downward, known as the descendens cervicalis. It joins with the descendens hypoglossi to form the ansa hypoglossi and provide the nerve supply for the extrinsic muscles of the larynx (fig. 2).

Plan of the Present Study.—The goal of the present study was to prove that the residual movements of the tongue in this instance of bilateral glossoplegia are passively produced by the movement of the hyoid bone, which, as described, is most probably effected by the geniohyoid muscles. It was assumed that demonstration of a forward movement of the hyoid bone resulting from the patient's attempt to protrude the tongue could be accepted as a proof of my hypothesis. For this purpose a special technic of roentgen examination was arranged. Two pictures were taken, the first with the tongue at rest and the second

7. Cunningham, D. J.: Text-book of Anatomy, ed. 2, New York, William Wood & Company, 1905, p. 697.

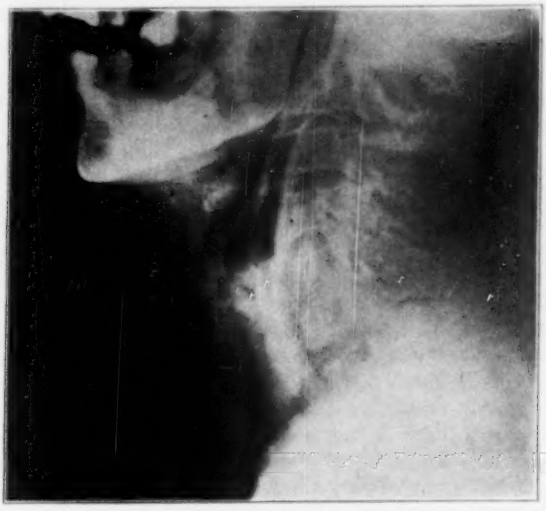

Fig. 4.—Superimposed double exposure in the reported case. Note that the distance between the two shadows of the vertebral columns measures 5 mm., while the distance between the two hyoid shadows is 9 mm.

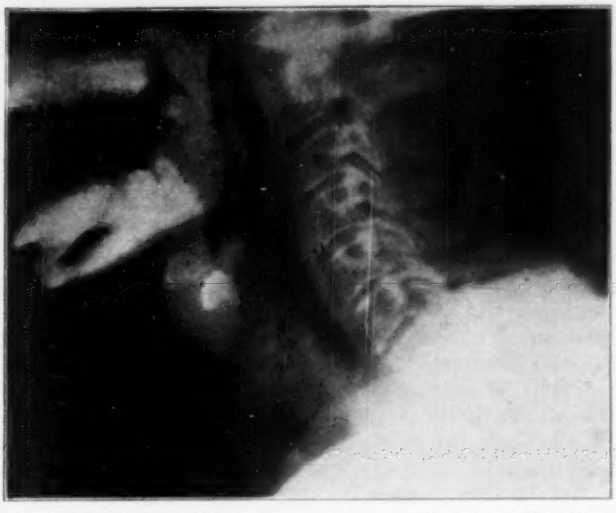

Fig. 5.—Superimposed double exposure on a normal subject. Note that the distance between the two shadows of the vertebral columns is 3 mm. while the distance between the two shadows of the hyoid bone is about 11 mm.

when the patient was asked to protrude the tongue as far as possible. The two pictures were taken superimposed one on the other, one film only for the two exposures being used. The patient, of course, was asked to keep as quiet as possible (fig. 4). Unfortunately, on account of her debilitated condition, the patient was unable to keep herself completely at rest during the time of these two exposures. One sees, however, the "two" hyoid bones are separated from each other by 9 mm., while the distance between the "two" vertebral columns responsible for the unavoidable movement of the head measures only 5 mm. Therefore the patient's attempt to project the tongue appears to be accompanied with a forward movement of the hyoid bone over a distance of 4 mm. Thus it is shown that the intraoral movement of the tongue is associated with and apparently caused passively by the pull on the hyoid bone, which, in turn, is produced by the action of muscles attached to it.

Figure 5 illustrates the situation when the same technic of superimposed double exposure was applied on a normal person. Even this volunteer was not able to keep entirely at rest during the two exposures. The distance between the "two" hyoids measured 11 mm., while the vertebral column moved only 3 mm. Therefore the true movement of the hyoid bone was 8 mm. Of course, under normal conditions both factors, the active movement by the hyoid muscles and the passive pull by the genioglossal muscles, are effective.

COMMENT

From these studies evidence is obtained that in a case of panglossoplegia the attempt to protrude the tongue is accompanied with forward movement of the hyoid bone. This movement, felt by palpation, is demonstrated objectively by a specially adapted roentgen technic. It is apparently caused by muscles which are not supplied by the hypoglossal nerve. The residual tongue movements, as noticed in the present case, are explained as being produced passively by means of the pull on the hyoid bone due to the action of the geniohyoids. Accordingly, two kinetic factors are to be regarded as responsible for the forward movements of the tongue, an active and a passive. The nerve supply for the former is provided by the twelfth cranial nerve; for the latter, by upper cervical nerves. These anatomic facts explain why in a case of panglossoplegia some forward movement of the tongue remains. Therefore the diagnosis of complete paralysis of both hypoglossal nerves cannot be contradicted by the presence of residual movements of the tongue. It must be left for future clinical studies to determine whether absence of residual movements of the tongue in instances of panglossoplegia can be regarded as a sign indicative of an extracranial location of the twelfth nerve lesions.

SUMMARY

In a case of complete panglossoplegia, paralysis of the orbicularis oris muscle and persistence of some voluntary forward movement of the tongue presented two problems which initiated these studies. The former is explained, according to Bing, by the fact that the nerve fibers responsible for the motor supply of this muscle originate mainly in the nucleus of the twelfth nerve. On the basis of this anatomic peculiarity paralysis of the orbicularis oris muscle associated with paralysis of the twelve nerve represents a sign indicative of nuclear location of the hypoglossal nerve lesion. It is assumed that on the same basis in cases of facial paralysis the selectively preserved action of this muscle can be of topognostic value in differentiating between nuclear and supranuclear lesions, on the one hand, and infranuclear lesions, on the other. In peripheral facial nerve lesions it may be particularly useful in topognosis of the nuclear versus the infrachordal locations of the lesion. The second problem is presented by the presence of some restricted forward movements of the tongue. These movements are explained and proved to be caused passively by the action of the geniohyoid muscles, which are not supplied by the twelfth cranial nerve. For this purpose a special roentgen technic, the "superimposed double exposure," was adapted. The movement of the hyoid bone caused by the attempt to move the tongue is demonstrated objectively. Therefore these movements of the tongue are to be regarded as passive; their presence does not contradict the diagnosis of complete paralysis of the twelfth nerve, but their absence may well be of topognostic value in differentiating extracranial lesions from intracranial lesions in instances of panglossoplegia.

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ORIGIN AND TREATMENT OF OSTEOMAS OF THE PARANASAL SINUSES

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MANY osteomas of the paranasal sinuses are not reported. Since 1930, however, 51 patients with such lesions have been encountered at the Mayo Clinic, a fact which would suggest that these tumors occur more commonly than would be supposed on the basis of reports in the literature. Although an osteoma of the frontal sinus was successfully treated by Veiga in 1506 and described in 1586,[1] the first recorded attempt at removal of such a lesion in the United States was not made until 1864, and in that instance the attempt ended in failure.[2] Generally, an osteoma of the paranasal sinuses is not reported until after it has attained such size that it is invading adjacent structures and producing symptoms so unmistakable that the need for surgical treatment is urgent.

No one, to our knowledge, has investigated the frequency with which evidence of osteomas, large or small, of the paranasal sinuses occurs in routine roentgenograms, but in view of Childrey's [3] study involving 3,510 roentgenograms of the sinuses, 0.43 per cent of which disclosed osteoma, we judge that the figure for large or small osteomas of the paranasal sinuses would be somewhat less than 0.43 per cent.

THEORIES AS TO CAUSATION

There is no agreement as to the cause of osteoma. Its origins have been said to be (1) embryologic, (2) traumatic and (3) infectious.

Theory of Embryologic Origin.—At the junction of the frontal and ethmoid bones two different types of bone, endochondral and membran-

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1. Teed, R. W.: Primary Osteomas of the Frontal Sinus, Arch. Otolaryng. **33**:255-292 (Feb.) 1941.

2. Gatewood, W. L., and Settel, N.: Osteoma of the Frontal Sinus: Review of the Literature and Report of a Case Presenting Extended Invasion of the Orbit, Arch. Otolaryng. **22**:154-164 (Aug.) 1935.

3. Childrey, J. H.: Osteoma of Sinuses, the Frontal and Sphenoid Bone: Report of Fifteen Cases, Arch. Otolaryng. **30**:63-72 (July) 1939.

ous, come into contact. Cohnheim pointed out that when in the course of development tissues of different natures come together growth of a tumor is likely to take place. In 1941, however, Teed [4] surveyed the literature and found that in only 26 of 128 cases of osteoma of the frontal sinus did the tumor develop in this region of junction. Of our own 40 cases of osteoma of the frontal sinus, in only 4 had the growth originated in this region.

Theory of Traumatic Origin.—It has been speculated that the more exposed life of the male results in the higher frequency of formation of osteomas among men. Smith [4] wrote that the essential primary process is one of healing and repair with formation of new bone and that the sequence of events would be trauma, giant cell tumor, bone cyst, osteitis fibrosa and osteoma. Yet, in spite of the great frequency of trauma in childhood, few osteomas occur in children. We did not have a patient in the first decade in our series, and a definite history of trauma was present in only 3 cases.

Theory of Infectious Origin.—Those who accept the theory of infectious origin feel that the great frequency of associated suppurative sinusitis is evidence in its favor. Ersner and Saltzman [5] have said that the presence of a low grade infecting organism having the opsonic selectivity for the stimulation of osteoblasts and causing an exudative process might result in a soft mass with an affinity for the deposition of calcium salts and thus form an osseous growth. But others [6] have declared that when infection is present, it is secondary to the deformity caused within the sinus by the osteoma. There was evidence of infection of the involved sinus in 12 of our cases.

Other Theories.—Theories such as those which involve ossified polyps, tuberculosis and syphilis have been largely discredited. In our series there was a positive reaction to serologic tests for syphilis in 2 cases and a history of syphilis in 1 case. Some unknown primary factor of a nutritional or metabolic nature may play an important part.

PATHOLOGIC ASPECTS

Although many authors have stated that there are three main pathologic types of osteoma—compact, spongy and mixed—the definition of this entire group of tumors is difficult.

4. Smith, A. T.: Osseous Lesions of Nose and Sinuses, with Special Reference to Hypertrophic Changes and Tumor Formations, Arch. Otolaryng. **31**:289-312 (Feb.) 1940.

5. Ersner, M. S., and Saltzman, M.: Osteoma of the Sinuses, Laryngoscope **48**:29-37 (Jan.) 1938.

6. Coates, G. M., and Krauss, F.: Osteoma of the Frontal Sinus, Ann. Otol., Rhin. & Laryng. **50**:450-457 (June) 1941.

Ewing [7] stated that circumscribed overgrowth of bone occurs under such a wide variety of conditions and the distinctions between inflammatory and neoplastic hyperplasia of the tissue are so often obscure that it has never been possible to define exactly the limits of osteoma. Davies-Colley [8] classified osteomas under the focal forms of fibrocystic disease of bone. He said that osteomas are part of a group of tumors comprising a wide range of neoplasms, beginning at one end with a tumor composed of pure fibrous tissue and reaching at the other a tumor composed of pure bone. Between these extremes are intermediate forms which illustrate every degree of proportionate admixture of the two substances.

Brunner and Spiesman [9] wrote that the mixed type of osteoma with a spongy core and a compact cortex is the most frequent in occurrence. They added that small osseous tumors which bulge into the sinuses and are covered by sinus mucosa actually are not neoplasms but are osseous hyperplastic processes which could be called "exostoses." They stated the belief that surgical removal is not necessary in these cases, since the lesions do not grow as do true osteomas.

The osteomas comprising our series were classified as such on the basis of either roentgenologic examination or microscopic study by a pathologist.

NATURE OF PRESENT STUDY

In our series of patients, 36 were male and 15 were female. The average age was 37.36 years, the youngest being 10 years old and the oldest 79 years. Five were in the decade 10 to 19 years; 11 were in the decade 20 to 29 years; 15 were in the decade 30 to 39 years; 12 were in the decade 40 to 49 years; 4 were in the decade 50 to 59 years; 3 were in the decade 60 to 69 years, and 1 was in the decade 70 to 79 years.

Generally, growth of an osteoma is very slow. We found that if symptoms and signs were present when the patient was examined the average duration of them was 3.7 years. Surgical removal was employed in 29 of our 51 cases, and in 22 cases the lesions were not disturbed but their course was followed subsequently by roentgenograms. In 40 of these cases the osteomas, as revealed roentgenologically or at operation, had originated in the frontal sinuses; in 9 cases, in the ethmoid sinuses, and in 2 cases, in the maxillary sinuses. In no case did an osteoma originate in the sphenoid sinuses.

7. Ewing, J.: Neoplastic Diseases: A Treatise on Tumors, ed. 4, Philadelphia, W. B. Saunders Company, 1940, pp. 219-223.

8. Davies-Colley, cited by Smith.[4]

9. Brunner, H., and Spiesman, I. G.: Osteoma of the Frontal and Ethmoid Sinuses, Ann. Otol., Rhin. & Laryng. **57**:714-737 (Sept.) 1948.

We shall next consider, in sequence, (1) osteomas of the frontal sinuses, (2) osteomas of the ethmoid sinuses and (3) osteomas of the maxillary sinuses.

OSTEOMAS OF THE FRONTAL SINUSES

Osteomas are said [10] to be the most frequently encountered neoplasms of the frontal sinuses. Of the 40 patients with osteomas of the frontal sinuses in this series, 29 were male and 11 were female. Surgical removal was done in 19 cases, with no deaths. Lesions not operated on either were too small or were accidentally found at roentgenologic examination and were asymptomatic. Some of the lesions are still being observed roentgenologically from time to time in an effort to determine their rate of growth.

Fig. 1.—(*a*) Osteoma arising from the posterior wall of the left frontal sinus; (*b*) same osteoma nine years later, showing an increase in size of approximately 30 per cent.

In figure 1 are shown roentgenograms of a patient who presented himself for examination at the age of 49 years. An irregular osteoma (fig. 1*a*) is seen to arise from the posterior wall of the left frontal sinus near the midline; nine years later (fig. 1*b*), it appears that there has been an increase of approximately 30 per cent in the size of the tumor. The patient had no symptoms directly referable to his osteoma and was advised only to have roentgenograms made periodically.

Such conservative management is in agreement with the views of Lillie,[10] who said it is not necessary to consider surgical removal of an osteoma of the frontal sinus unless definite symptoms and signs seem

10. Lillie, H. I.: External Operations on the Frontal Sinus, Am. J. Surg. **42**:199-206 (Oct.) 1938.

to demand such treatment. Hempstead,[11] and Brunner and Spiesman [9] have expressed the belief that any evidence of expansion of an osteoma is an indication for removal. Teed [1] declared that operation should be carried out while the tumor is small rather than after the appearance of symptoms of extrasinusal extension. Beck [12] and Jeschek,[13] however, reported that when patients with osteomas of the frontal sinus are not operated on the mortality rate may be as high as 48 per cent as a result of complications and that in all cases, excepting the rare instances of spontaneous necrosis and extrusion of the tumor, radical operation must be performed.

Osteomas lying loose in the sinus have been noted, but it is rather doubtful that this occurs spontaneously. Such tumors probably were attached to the anterior wall, and the first blow on the surgeon's chisel tended to break the pedicle and to make the tumor appear loose when it was finally exposed.

TABLE 1.—*Extension of Osteomas of the Frontal Sinus Occurring Singly or in Conjunction in 27 Cases*

Extension or Perforation into	Times Noted
Ethmoid sinuses	9
(Bulging floor into) orbit	6
Anterior wall	4
Opposite frontal sinus	4
Posterior wall	2
Nasal cavity	1
Antrum	1

In 27 cases in our series osteomas had extended into neighboring structures as determined on the basis of roentgenologic or surgical evidence and as indicated in table 1. In most cases the tumor seemed to bulge or perforate into the orbit and to extend into the ethmoid sinuses on the same side. The latter is not a formidable obstacle when it is discovered at operation, since under such conditions the patients already have a large epithelium-line frontal duct.

Symptoms and signs in a large measure were dependent on size and extension of the tumor. The commonest symptom was frontal pain, noted in 10 cases. The next most frequent symptoms were profuse nasal discharge on the same side (noted in 7 cases) and periorbital swelling (noted in 7 cases) which caused displacement of the eyeball with diplopia in 4 cases.

11. Hempstead, B. E.: Osteomas of Paranasal Sinuses and the Mastoid Process, J. A. M. A. **111**:1273-1275 (Oct. 1) 1938.

12. Beck, J. C.: Some Uncommon Types of Neoplasms About the Head: Osteoidoma of Frontal Sinus; Multiple Myeloma; Intrapalatal Mixed Tumor, Tr. Am. Laryng., Rhin. & Otol. Soc. **36**:374-386, 1930.

13. Jeschek, J.: Behavior of Frontal Sinus in Osteoma, abstracted, Arch. Otolaryng. **22**:126 (July) 1935.

Those patients with purulent nasal discharge had an infection of the frontal sinus as a complicating feature. Such infection could nearly always be determined roentgenologically on the basis of evidence of retained secretion, thickened mucous membrane or both.

One patient complained of pain over the right frontal area. A roentgenogram of the sinus revealed an osteoma the size of a hazelnut. Removal of the osteoma gave complete relief from pain.

Two patients had a draining fistula in the eyebrow which was caused by incomplete removal of the osteoma, performed elsewhere. One patient had symptoms and signs of involvement of the central nervous system. In 20 cases osteomas of the frontal sinuses gave no symptoms.

The following conditions were found at operation: retained pus or granulation tissue, 8 cases; retained mucus, 4 cases; fistulas in the eyebrows, 2 cases; pyocele, 1 case; brain abscess, 1 case, and pneumoencephalocele, 1 case.

Retained mucus probably is present in most frontal sinuses when the osteoma is large and blocks the frontal duct; this mucus almost always is sterile. The absence of mucoceles in our series was rather unusual, since there are several reports in the literature that a mucocele was found in conjunction with an osteoma.

A case involving an associated pneumoencephalocele was most interesting and deserves further comment.

ILLUSTRATIVE CASE 1.—A man 26 years old was seen at the clinic on Feb. 28, 1948. He had been in perfect health until eight months previous to his admission, when severe headaches developed that were aggravated by blowing the nose. The headaches were located at the base of the skull and extended to the vertex and forehead and were severer on motion. One month before coming to the clinic this patient was confused mentally and had some degree of amnesia but no headache at the time. Ten days before his admission, some degree of weakness in the left arm developed. Two days later he had a staggering gait. At no time was there any history of rhinorrhea. When the patient was examined, a slight protuberance of the right frontal area was noted. Otherwise, results of examination of the ears, nose and throat were essentially negative. Neurologic examination revealed hyperactive tendon reflexes of the left arm and leg, with weakness. Roentgenograms disclosed a large osteoma that filled the right frontal sinus and extended across to the left side. The anterior wall of the frontal sinus was expanded and thin. The posterior wall was irregularly calcified; this probably represented perforation. A large subdural collection of air was present in the right frontoparietal area (fig. 2*a* and *b*). The electroencephalogram demonstrated localization of delta activity in the right frontal area. The patient was operated on by an otolaryngologist and a neurosurgeon. After removal of the frontal bone the patient was found to have a chronic abscess of the brain on the right side of the frontal area; the cavity of the abscess had filled with air, forming a pneumatocele. It was thought that the osteoma probably had grown and penetrated the dura and tip of the frontal lobe, so that infectious material together with air from the sinus had entered the subdural space, especially when the patient coughed, blew his nose

or sneezed. The dural opening was closed and 5 cc. of penicillin was then left in the cavity. The external table of bone was removed from the right frontal sinus, and the osteoma was "rocked" out. The mucosa of the sinus was entirely removed. The bone flap was replaced with wire sutures, and the scalp wound was closed. The patient made an uneventful recovery and was dismissed from the hospital after two weeks. Reexamination a year later disclosed that the patient had remained well.

A number of writers[14] have reported cases of pneumatocele secondary to osteomas of the frontal or ethmoid sinus.

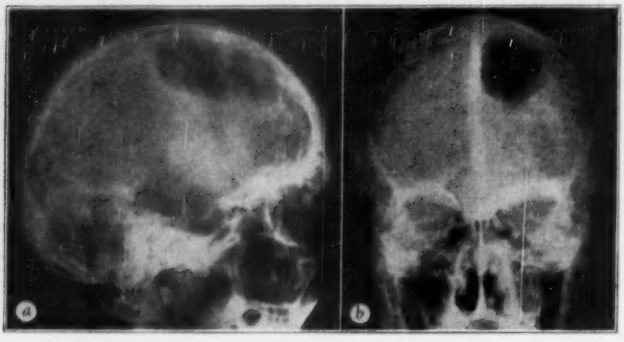


Fig. 2.—Large pneumoencephalocele (case 2) secondary to an osteoma of the right frontal sinus which had perforated the posterior wall.

14. Cushing, H.: Experiences with Orbito-Ethmoidal Osteomata Having Intracranial Complications, with Report of Four Cases, Surg., Gynec. & Obst. **44**:721-742 (June) 1927. Armitage, G.: Osteoma of the Frontal Sinus, with Particular Reference to Its Intracranial Complications, and with the Report of a Case, Brit. J. Surg. **18**:565-580 (April) 1931. Bell, F. G.: Intracranial Pneumatocele (Pneumocephalus) Associated with an Orbito-Ethmoidal Osteoma, Australian & New Zealand J. Surg. **4**:70-74 (July) 1934. Baldenweck, L., Mallet, Thévenard and Jouveau-Dubreuil: Pneumatocèle frontale interne au cours de l'évolution d'un ostéome du sinus frontal, Ann. d'oto-laryng., 1934, pp. 657-668. Nowotny, K., and Schüller, A.: Subduraler Pneumokephalus bei ethmoidalem Osteom (Status epilepticus letalis nach lumbaler Luftfüllung bei altem Morbus sacer), Röntgenpraxis **8**:107-108 (Feb.) 1936. Benjamins, C. E., and Verbeek, F. A. L. J.: Pneumatocèle frontale interne et ostéome du sinus frontal, Ann. d'oto-laryng., 1936, pp. 881-892. Devic, Ricard and Moreau: A propos de deux cas d'arachnoïdite spinale haute: Considérations cliniques et thérapeutiques, Lyon méd. **161**:723-730 (June 19) 1938. Campbell, E. H., and Gottschalk, R. B.: Osteoma of Frontal Sinus and Penetration of Lateral Ventricle, with Intermittent Pneumocephalus, J. A. M. A. **111**:239-241 (July 16) 1938. Kessel, F. K.: Orbito-Ethmoidal Osteomata with Intracranial Complications: Report of a Case, Guy's Hosp. Rep. **89**:337-346, 1939. Brunner and Spiesman.[9]

Another case deserves special mention, since no similar case has been reported so far as we could find.

ILLUSTRATIVE CASE 2.—A 29 year old man, who came to the clinic on Sept. 12, 1944, gave a history of epiphora of the left eye and also of pain on moving the eye to the left. He also had had fever in which the temperature ranged up to 101 F. (38.3 C.), with swelling around the eye. All these difficulties subsided after the use of chemotherapy. Roentgenograms of the sinuses disclosed a large osteoma that filled the left frontal sinus and extended into the right frontal sinus and down into the left ethmoid sinus. The patient was operated on Sept. 14, 1944. A coronal incision was employed because of the large size of the tumor and also because there was evidence of infection (much pus came from the left side of the nose). The osteoma was about 2 by 4 cm. and looked more or less like any other osteoma. Microscopic examination revealed most of the tissue to be osteoma, but scattered through it were areas of osteogenic sarcoma, grade 2, representing sarcomatous degeneration of the osteoma. When this discovery was made, it was decided not to reopen the area but rather to use electrocautery, because (1) the degree of malignancy was low, (2) the degenerated area was thoroughly removed and (3) the origin of the osteoma was in the region of the cribriform plate.

As mentioned previously, of the 40 osteomas of the frontal sinus, 19 were removed surgically, with no postoperative deaths. In all operations, general anesthesia usually was produced intratracheally; regional infiltration for hemostasis was used except for three primary operations and one secondary operation, in each of which topical anesthesia plus infiltration was used.

The incision varied somewhat, but in 17 cases it was made through the eyebrow. In 2 cases in which the tumor was very large and extended to the contralateral side, a coronal incision was used. In 2 cases it was necessary to utilize an additional incision, which extended through the canine fossa into the antrum. In 17 cases the osteoma was removed, with preservation of as much of the wall of the sinus as possible. In 2 cases the frontal sinus was ablated.

In 8 cases in which the frontal sinus was preserved and the nasofrontal duct was not disturbed there was no difficulty with drainage or closure of the duct. In 8 cases in which the frontal sinus was preserved, the nasofrontal duct was enlarged and a petrolatum pack, rubber catheter or polyethylene tube was left in place at the time of operation, there were 2 cases in which one subsequent operation was necessary and 1 case in which two subsequent operations were necessary because of closure of the nasofrontal duct. This experience indicates that whenever possible the frontal duct should be left intact, a view shared by Dowling.[15] There are cases in which the osteoma is associated with severe chronic disease of the frontal sinus. In most of such cases there

15. Dowling, J. R.: Osteoma of the Frontal Sinus: Report of Five Cases, Arch. Otolaryng. **41**:99-108 (Feb.) 1945.

is justification for enlarging the nasofrontal duct in an effort to establish adequate drainage. Although our follow-up study was incomplete, we did not record any instance of recurrence of an osteoma.

OSTEOMAS OF THE ETHMOID SINUSES

In this series there were 9 cases of osteoma of the ethmoid sinuses. Six patients were male, and 3 were female. The average age of a patient at the time of examination was about 26 years, as compared to an average age of 39 years for the patients with osteoma of the frontal sinus. The youngest patient was 10 years old; the oldest, 62. As determined roentgenologically or surgically, 1 of the tumors was attached to the lamina papyracea and 1 was located in, or attached to the walls of, the anterior ethmoid cells. The site of attachment of 7 osteomas could not be determined.

TABLE 2.—*Extension of Osteomas of Ethmoid Sinuses Occurring Singly or in Conjunction in 9 Cases*

Extension or Perforation into	Times Noted
Frontal sinus of same side	4
Antrum	4
(Bulging wall into) orbit	3
Nasal cavity	3
Roof of orbit	1
Extending into both frontal sinuses, perforating anterior wall of frontal sinus on same side	1
Sphenoid sinus	1

As would be expected, these tumors involve neighboring structures early. It has been said [10] that the tumor often enters every individual ethmoid cell; the extensions may be compared to the progress that an ameba makes by pseudopodia. In this series, extension occurred in all 9 cases, as shown in table 2.

Because of the relatively restricted space of the ethmoid sinus, an osteoma situated there will produce symptoms earlier than will an osteoma of the frontal sinus. In our series the average duration of symptoms of an osteoma of the frontal sinus was 3.7 years; the average duration of an osteoma of the ethmoid sinus was 1.6 years. There were no "silent osteomas" in the group of osteomas of the ethmoid sinuses. Frontal or maxillary pain was the commonest symptom; it occurred in 6 cases. Early symptoms also were nasal obstruction on the same side (3 cases) and a palpable mass in the orbit (3 cases). In 2 cases there was profuse nasal discharge on the same side; in 2 there was displacement of the eyeball, and in 1 there was diplopia. In 1 case symptoms of intracranial involvement occurred. In all cases the diagnosis was made by examination with roentgen rays. A point in differential diagnosis is that a meningioma resembles an osteoma except that roentgenologically a meningioma is less dense.[10]

In 4 cases infection was also present, and in 1 case a brain abscess occurred. Surgical removal of the osteoma was done in 8 cases. Submucous resection was done in 1 case as a minor preoperative surgical procedure, because the nasal septum was deviated markedly to the side of the lesion. In 1 case surgical treatment was not employed. The patient in question had, in addition to an extremely large osteoma that occupied a considerable part of the base of the skull, two obtuse osteomas elsewhere in the cranium.

In all cases regional anesthesia for hemostasis and intratracheal anesthesia were used. In all cases the operation started as an externl frontoethmoid incision; in 1 case it was necessary to utilize an additional incision that extended through the canine fossa into the antrum. Different approaches have been advocated, but any type of operation may be employed so long as it exposes the field adequately.

Of those patients whose predominant symptom was frontal or preorbital pain, 4 obtained relief. In 1 case there was no relief, and in another case pain due to closure of the nasofrontal duct recurred but disappeared after the second operation. In 3 cases the nasofrontal duct was enlarged and a rubber tube was left in place. In 2 of these cases reoperation was done, one and nine years postoperatively, because of closure of the nasofrontal duct. The lesson, again, is that whenever possible the frontal duct should be left intact.

The complications of surgery are said to be injuries to large blood vessels, meningitis, brain abscess, atrophy of an optic nerve, loss of the eyeball and deformities.[12] In our series of 8 patients operated on for osteoma of the ethmoid, 1 died. The condition of this patient deserves further mention.

Illustrative Case 3.—A woman 33 years old was first seen at the Mayo Clinic on Oct. 1, 1946. Seven months previous to her admission she had had frontal headaches, with fever which persisted for about three weeks. This trouble seemed to clear up after sulfonamide therapy. Three months before coming to the clinic the patient had had a similar attack. Examination at the time of her admission revealed a hard tumor in the right side of the nose; the tumor also could be seen in the nasopharynx. Roentgenograms disclosed a hard, lobulated osteoma that partially filled the right nasal fossa and completely filled the right ethmoid cells and the lower portion of the right frontal sinus, with apparent extension into the medial portion of the right antrum and the right sphenoid sinus. There was also some retained secretion in the right antrum. Two incisions were made. The first was the regular frontoethmoid incision through the eyebrow; the second was made through the right canine fossa. On removal of the osteoma from the roof of the orbit there was profuse flow of 1 to 2 fluid ounces (30 to 60 cc.) of thick pus from a perforation in the dura. A definite stalk of a brain abscess was seen. The patient died two days later. Necropsy revealed a drained abscess of the right frontal lobe; a second abscess was found between the falx cerebri and the frontal lobe. There was much edema of the brain.

OSTEOMAS OF THE MAXILLARY SINUSES

True osteomas of the maxillary sinus are rare; Rawlins [16] was able to collect only 30 cases from the literature and has observed 3 such lesions personally. In our series there were 2 cases of osteoma of the maxillary sinus; both patients were male. Also encountered in our study, but not included in the present report, were 23 cases of osteoma or osteofibroma of the maxilla which involved or bulged into the antrum secondarily. They all seemed to have originated in or on the anterior wall of the maxilla and to have caused early deformity of the face. In these cases, only partial removal was carried out, for cosmetic reasons.

Of the 2 patients with true osteoma of the antrum, 1 gave a history of trauma. As determined at operation, in 1 case the tumor was attached on the medial wall along the margin of the inferior meatus, and in the other it was attached to the lateral wall by a pedicle. Symptoms were indefinite; occasional pain over the involved side was reported to have persisted for a year or two. As in the case of the other osteomas, the diagnosis of osteoma of the antrum was based on roentgenologic evidence. In addition to the tumor, the roentgenograms showed thickened mucous membrane. In both cases the osteoma was removed by the Caldwell approach, and a large window was made in the antrum underneath the inferior turbinate process. Both patients were relieved of their symptoms, and there were no recurrences.

No cases of primary osteoma of the sphenoid sinus were recorded in the clinic files. Survey of the literature did not disclose any report of a case of proved osteoma originating in this sinus.

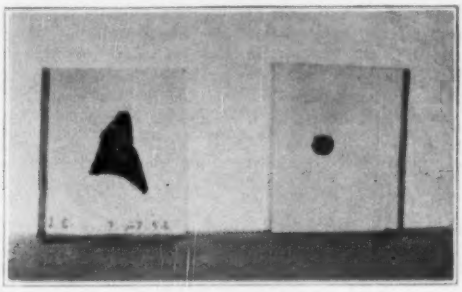
16. Rawlins, A. G.: Osteoma of the Maxillary Sinus: Report of a Case, Arch. Otolaryng. **32**:499-505 (Sept.) 1940.

Clinical Notes; New Instruments and Technics

A METHOD OF PRESERVING FOREIGN BODY SPECIMENS RECOVERED BY ENDOSCOPY

ERNEST B. EMERSON Jr., M.D.
ROCHESTER, N. Y.

IN THE past, one of the minor problems in the work of the endoscopist with foreign bodies has been preservation of the specimens. No suitable mediums had been found in which to mount in collection form all the different specimens encountered. Pins, coins and other such objects were easily saved in their natural state, but the convenient handling of such widely differing specimens as a raw oyster or peanut required special technics and, often, unbreakable specimen containers.



A chicken bone and a garnet bead mounted in plastic. The specimen on the left has been permanently inscribed.

Recently, in this department, we have been using "bio-plastic," manufactured by Ward's Natural Science Establishment, Inc.[1] The results have been highly satisfactory. The specimens mounted range from chicken bones to fresh shrimp. Such specimens as peanuts retain their full shape, and colored objects retain the color seen at operation.

In addition to preserving the specimen, the plastic can be inscribed to preserve the record of the case, thereby eliminating one more hazard of the older methods, separation of the label from the specimen.

The plastic, when fully hardened, is nearly indestructible, this quality permitting safe handling by students or lecture audiences.

From the Department of Surgery, Division of Otolaryngology, University of Rochester School of Medicine and Dentistry.

1. "Bio-plastic" and authoritative data on it may be obtained from Ward's Natural Science Establishment, Inc., P. O. Box 24, Beechwood Station, Rochester, N. Y.

TECHNIC

For such objects as pins, coins and beads no special preparation of the specimen is needed. However, for foreign bodies such as peanuts, the specimen should be allowed to dry in room air until, with an ordinary magnifying lens, the surface first begins to show crinkling. Fleshy specimens such as shrimp or a bolus of meat are best prepared first by fixing in formaldehyde, then removing the excess fixative by washing with water and removing the surface water by blotting.

When the specimens are ready, a layer of "bio-plastic" is poured into a mold (a Petri dish or ordinary icebox dish is satisfactory, the size depending on the number of specimens to be mounted) and is allowed to solidify at room temperature until nearly hard. The specimens are then planted on this surface, and a second layer of the "bio-plastic," equal in thickness to the first layer, is poured over them. This second layer is allowed to set until the plastic does not tend to flow when the mold is tipped. A piece of glass is then placed over the surface of the second layer, carefully, in order not to trap air beneath it; this maneuver prevents contamination of the surface during the rest of the process and, in combination with the glass bottom of the mold, gives the finished plastic block two clear surfaces, which do not need additional polishing.

The whole preparation is then allowed to remain at room temperature for twenty-four hours. At the end of this time the mold is placed in a constant temperature oven. The oven temperature should be 38 C. at the start and should be slowly raised to 60 C. and kept at that point for one to two hours. At the end of this time the oven is opened and the preparation allowed to cool slowly.

The whole block is then removed from the mold by mere tapping of the bottom, as since in the process of setting the block shrinks a little from the sides of the container.

The block of plastic may then be cut to any desired shape with any fine saw and the cut surfaces polished, if it seems desirable, or left opaque, as in the illustration.

Data pertinent to the particular specimen are then easily inscribed, on either the polished or the opaque surface of the individual specimen blocks. This procedure is easily accomplished with a vibrotool or any high speed hand burr.

26 Strathallan Park.

News and Comment

HOME STUDY COURSES OF THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The 1950-1951 home study courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, Executive Secretary-Treasurer, 100 First Avenue Building, Rochester, Minn.

MEETING OF THE OTOLARYNGOLOGICAL SOCIETY OF ISRAEL

The annual meeting of the Otolaryngological Society of Israel was held on Jan. 26-28, 1950 in Tel-Aviv. The first discussion of the meeting was devoted to the problem of the hard of hearing. Dr. Sherman, of Tel-Aviv, and Dr. Lachmann, of Jerusalem, read papers of introduction, and Dr. Sadowsky and Dr. Schwarzbart, of Tel-Aviv, Dr. Höxter, of Jerusalem, and Dr. Zaliuk, of Haifa, participated in the discussion. On the second day of the meeting Dr. Wodak, of Tel-Aviv, presented a report entitled "New Views about the Vestibular Apparatus." Dr. Sherman was elected president and Dr. Wodak honorary secretary of the society.

REVISED EDITION OF "REVIEWS OF MEDICAL MOTION PICTURES" NOW AVAILABLE

The Committee on Medical Motion Pictures of the American Medical Association has completed the second revised edition of the booklet entitled "Reviews of Medical Motion Pictures." This booklet now contains 225 reviews of medical and health films reviewed in *The Journal of the American Medical Association* to Jan. 1, 1950. Each film has been indexed according to subject matter. The purpose of these reviews is to provide a brief description and an evaluation of motion pictures which are available to the medical profession. Each film is reviewed by competent authorities, and every effort has been made to publish frank, unbiased comments. Copies are available at a cost of 25 cents each from Order Department, American Medical Association, 535 North Dearborn Street, Chicago 10.

Abstracts from Current Literature

Ear

SOME OBSERVATIONS UPON THE OTOLOGICAL EFFECTS OF STREPTOMYCIN INTOXICATION. M. R. DIX, C. S. HALLPIKE and M. SPENCER HARRISON, Brain **72** (pt. 2):241, 1949.

This article deals with the genesis of the fast component of nystagmus. The authors cite the theories that its origin is in the cerebrum or, according to Lorente de Nó, in the reticular formation. Of particular interest is the discussion of the theory of Ohm and Ter Braak. Ohm stressed the fact that because vestibular and optokinetic nystagmus both exhibit an identical rhythmic alternation of slow and rapid components they both must depend on some common, anatomic mechanism. To support this opinion, he demonstrated that when the retina as well as the labyrinth is stimulated to produce a nystagmus the result is a smooth summation of the optokinetic as well as the labyrinthine elements. Dix, Hallpike and Harrison set out by assuming that if the rapid component of the ocular nystagmus has an origin similar to that of the vestibular nystagmus both of them should be abolished by the use of such drugs as streptomycin. The authors investigated a series of 12 patients who were treated with streptomycin. They applied the galvanic test as well as the alternate hot and cold caloric test of Fitzgerald and Hallpike as a means of determining the status of the inner ear and vestibular nuclei. They found that the galvanic reactions were in no way reduced by the administration of streptomycin in all except 1, in which, they believe, the streptomycin was so effective in destroying the vestibular nuclei that not even the galvanic response was present. However, in 7 of the 12 patients given the caloric test there was no response at all; in 4 it was very much reduced, and in 1 the response was normal. From their findings, they concluded that Ohm's and Ter Braack's idea is not tenable and that the central pathways of the optokinetic nystagmus are entirely independent of the vestibular nuclei.

SHUSTER, Philadelphia.

CLINICAL STUDIES WITH PARENTERAL VITAMIN A THERAPY IN DEAFNESS. M. J. LOBEL, Eye, Ear, Nose & Throat Monthly **28**:213 (May) 1949.

The author presents a group of approximately 300 patients with progressive deafness of all types, treated with an injectable vitamin A preparation. Lobel claims appreciable gain in hearing of hard of hearing patients treated over a prolonged period, i. e., for more than five months. In many patients, tinnitus was relieved in less than two months of therapy. The response is more pronounced in the catarrhal form of deafness, whereas in the perceptive type of deafness the response is slow; but even in deafness of this type a favorable change may take place.

JENNES, Waterbury, Conn.

A NEW TREATMENT FOR VERTIGO. L. A. WITZEMAN, Eye, Ear, Nose & Throat Monthly **28**:272 (June) 1949.

The author reports the use of dimenhydrinate ("dramamine") for the symptomatic complaint of vertigo in 47 patients, with complete relief in all but 4 and with improvement in 2 of these. Witzeman used the drug in those cases in which

the vestibular apparatus was the probable seat of the disturbance. No patient in this group had Ménière's syndrome. The author considers the drug as a palliative agent affording the patient relief while the various etiologic factors are being determined or in cases in which the cause cannot be found.

JENNES, Waterbury, Conn.

WILL NOISE DAMAGE ONE'S HEARING? W. E. GROVE, J. A. M. A. **140**:674 (June 25) 1949.

This distinguished otolaryngologist reviews the manifold phases of noise as it is encountered in industry and in daily life and discusses how it produces fatigue, menaces health and lowers efficiency.

Other factors are considered besides the intensity of noise, and several figures are included which indicate the audiometric levels of loss of hearing of persons after they spent many hours working in a chipping school.

No form of treatment is effective once the lesion occurs in the neuroepithelium of the organ of Corti. Prevention of the lesion is the only means of attack; sounds, therefore, of injurious intensity must be prevented from reaching the ear. Such a procedure must be concerned with education and propaganda, and sporadic attempts to solve this problem, especially in airplane factories during the war, already have been made.

A subcommittee on industrial noise to the committee on conservation of hearing of the Academy of Ophthalmology and Otolaryngology, has been busily engaged during the past year in acquainting insurance carriers and independent employers of labor with the program, and has already succeeded in briefing labor and management in the problems involved. Appropriate methods of procedure have been worked out, and coordinative plans for the redesigning of buildings and the cooperation of the various departments with the otologist are now well under way.

Industrialists and farsighted businessmen are beginning to realize that industrial noise is a health hazard.

GORDON, Philadelphia.

HEARING CAPACITY OF THE NEWBORN INFANT. D. DE FRANCESCO, Lattante **19**:265 (May) 1948.

Hearing tests were made on 30 newborn infants; only 1 of these infants failed to show a reaction to noise sometime during the first five days of life. When sounds of wavelengths 512, 2,048 and 4,096 were produced by tuning forks or a Galton whistle, the infant would respond by some simple reflex motion; however, there was no response to sound of wavelengths 128 or 256. When the Lombard-Bárány noise apparatus was used, the infants showed a more complex and interesting reaction, which the author describes as a mimicking expression of the face; this reaction seemed to be more than a pure reflex, suggesting a conscious, although primitive, activity.

HIGGINS, Boston [AM. J. DIS. CHILD.]

SURGICAL TREATMENT OF OTOSCLEROSIS. MAURICE SOURDILLE, Acta oto-laryng. **37**:195 (June) 1949.

Sourdille states that surgical treatment of otosclerosis is based on two essential factors: establishment of a permanent labyrinthine fistula and mobilization of its membranous covering to create a new tympanic system. It is his belief that there has been no really decisive principle added to that suggested by him in 1935. No

matter what instruments have been used for the creation of the labyrinthine fistula —burr, gouge, chisel, curet; and whatever may have been the dimensions, the position, the form, the treatment of the osseous section—mechanical, chemical, physical, elimination of the osteal and endosteal dust, the qualities of the membranous covering, a decisive advance has really not been represented. He states that 50 per cent is the usual rate of cases with success. He believes that the only practical way as yet devised to increase the rate of success is to reopen the closed fistula and that the surgical technic should be developed with this procedure in mind.

Sourdille states the opinion that the incus must be conserved and that the head of the malleus be removed. Removal of the incus is indicated only in cases of slight deafness. Leaving the incus in position serves to protect the facial nerve. Conservation of the incus has the additional value of aiding in the conduction of the higher sound frequencies. He points out, however, that the conservation of the incus is of value only if the operation is done in two stages, in which the incus may be held in a cutaneous and cicatricial flap, thus giving the incus maximum mobility.

Sourdille states that agreement is needed between the different fenestrators and suggests that they work together at some common geographic point. He feels that specialization on the ear alone is desirable and that the specialty is now too broad.

ROBERT LEWY, Chicago.

Pharynx

ROENTGEN TREATMENT OF WALDEYER'S TONSILLAR RING IN CHILDREN. D. CIRILLO, Lattante **19**:327 (June) 1948.

Over a period of twelve years, roentgen irradiation of the tonsillar tissue was given to 435 children; follow-up reports were obtained on 206 of them. Favorable therapeutic results followed the irradiation in 90 per cent, and no benefit was noted in 10 per cent of the children. In no child was there any injury referable to the treatment.

HIGGINS, Boston [AM. J. DIS. CHILD.].

TONSILLECTOMY IN DIPHTHERIA CARRIERS. K. GRAF, Schweiz. med. Wchnschr. **78**:75 (Jan. 31) 1948.

The conservative treatment of the diphtheria carrier has not been followed by the desired results. The author is of the opinion that persistence of diphtheria bacilli depends on the presence of the disease in the mouth and nasal cavities, and that tonsillectomy and removal of the adenoids, when properly performed, nearly always eliminate the organisms. He also maintains that in cases in which the patient has been ill with diphtheria and the bacilli have not disappeared by the eighth week following recovery from the disease the operation may be performed. He recognizes that before operation conservative methods may be tried to eliminate the bacilli. He mentions various antiseptics, especially penicillin. He recommends that penicillin be used through injection or as a nasal inhalation or spray. If these methods do not succeed after three to four weeks' trial, operation should be resorted to. He is of the view that while the organisms may persist for a number of days after the operation, in most cases fourteen days after removal of the tonsils and adenoids bacilli are no longer found by cultural methods.

ART and ART, Chicago [AM. J. DIS. CHILD.].

Larynx

ROENTGEN THERAPY FOR SUBGLOTTIC LARYNGITIS IN CHILDREN. J. MARINONI and E. FRACASSO, Clin. pediat. **30**:247 (April) 1948.

Thirteen children with subglottic laryngitis were treated by roentgen irradiation. Excellent results were obtained in 2 of the 3 children who still showed constriction two to three weeks following tracheotomy. Good results were obtained with the treatment of 5 children with acute laryngitis (1 with the severe disease and 4 with the mild form). Only 1 of 4 children with subacute laryngitis obtained any relief from the treatments, and 1 child having the chronic disease with acute exacerbations was not benefited. It was apparent that the earlier in the disease the roentgen treatment was given, the better the chance for relief of the condition.

No serious difficulty was encountered from an immediate edema of the larynx following the irradiation; however, small initial doses are recommended for children without tracheotomy tubes. HIGGINS, Boston [AM. J. DIS. CHILD.].

Nose

MUCOCELE OF THE FRONTAL SINUS: SOME NOTES ON ITS AETIOLOGY, PATHOLOGY AND DIAGNOSIS. EDWARD F. STEWART, J. Laryngol. & Otol. **63**:70 (Feb.) 1949.

Mucocele of the frontal sinus, usually unilateral, is an indolent, insidious, slow, progressive dilation of the frontal sinus, at the site of an old infection and resulting from an accumulation of mucoid material. Stewart discusses the problems that arise when secondary infections supervene on an old chronic sinusitis. The theories on the production of mucoceles are enumerated. Because of their slow development, pain is not a predominant symptom in early cases. However, when an acute infection develops, pain is severe. Surgical intervention by relieving pressure and establishing drainage gives much relief. Several cases are reported, and the operative procedure is given in detail. LEJEUNE, New Orleans, La.

THE REMOVAL OF SECRETIONS FROM THE NASAL ANTRUM: THE PART PLAYED BY PRESSURE WHEN IRRIGATING THE NASAL PASSAGE WITH WARM SALINE SOLUTION; A PHYSICAL HYPOTHESIS. J. J. CANTOR, Laryngoscope **58**:1307 (Dec.) 1948.

Cantor attempts to prove theoretically how secretions may be removed from a sinus by irrigating the nasal cavity with warm sodium chloride solution. He shows that the warming, and hence expansion, of the air of the sinus, forces the secretions out, rather than that the fluid passes out by the ostium of the sinus. If the air is allowed to cool, repetition of the procedure will remove further secretions. He suggests that the procedure be called "the interrupted replacement method."

HITSCHLER, Philadelphia.

CONGENITAL ATRESIA OF THE NASOLACHRYMAL DUCT. NICHOLAS W. WOYWITKA, Canad. M. A. J. **59**:357 (Oct.) 1948.

A geneologic tree showing the occurrence of congenital atresia of the nasolacrimal duct over five generations is presented. Nine of 46 members of the family were afflicted. The question of penetrance of the dominant gene is commented on. McGUIGAN, Evanston, Ill. [AM. J. DIS. CHILD.].

Miscellaneous

CONGENITAL ATRESIA AND TRACHEOESOPHAGEAL FISTULA. WILLIAM P. LONGMIRE JR., Arch. Surg. **55**:330 (Sept.) 1947.

This congenital anomaly, if untreated, is incompatible with life. In recent years it has been satisfactorily corrected by surgical procedures. The author reports on 5 infants with congenital atresia of the esophagus and tracheoesophageal fistula who were operated on. In all instances the fistula was ligated and an end to end anastomosis of the esophagus performed. The first patient died immediately after operation. In the last 4 consecutive children the operations have been successful. These children are taking all feedings by mouth and are gaining weight satisfactorily. Certain features of the diagnosis, the operative technic and the postoperative care are discussed.

KAISER, Rochester, N. Y. [AM. J. DIS. CHILD.].

INFANT SPEECH: DEVELOPMENT OF VOWEL SOUNDS. ORVIS C. IRWIN, J. Speech & Hearing Disorders **13**:31-34 (March) 1948.

Analysis of the percentage of total occurrence of vowel sounds in each of fifteen age levels at two month intervals shows that while most of the vowel sounds are used by young infants, only three of them, the front vowel sounds *e* as in "set" and *i* as in "sit" and the middle vowel sound ʌ as in "sun," are consistently used by infants at the first age level of 1 to 2 months. The newborn infant's vowel sound repertoire is decidedly unlike that of the adult. There is an infant vocalization or preponderance of front and middle vowel utterance during the first year, which thereafter gradually diminishes, while back vowels increase, until at 2½ years the percentage distribution approximates that of the adult, without, however, being identical with it.

PALMER, Wichita, Kan.

THE SPEECH CORRECTIONIST ON THE CEREBRAL PALSY TEAM. JOHN C. SNIDECOR, J. Speech & Hearing Disorders **13**:67 (March) 1948.

The child with cerebral paralysis presents complications above and beyond articulatory deviations. The child, for example, may be aphasic or hard of hearing or may stutter. There has been almost no objective research related to speech training for children with cerebral paralysis. Each type of cerebral paralysis requires a different clinical approach. Achievement of relaxation, the cornerstone of all speech work, is the first step; easy, onflowing vocalization with relaxation is the next; exercise on individual sounds follows this. Simple nonsense syllables and words are then taught; more difficult words and sentences come next; then transfer of learning must be accomplished before the speech correctionist's job is completed. The child is taught to speak, still relaxed, in a sitting, and then in a standing, position. The mental hygiene aspects of speech correction should be kept in mind at all times. Frequently children think they will learn to speak effectively in a short time; but emphasis must be placed on the pleasure arising from the task, and on small gains, in order to avoid disappointment from expectations of improvement in a stated period. The child with cerebral paralysis needs warmth and affection. The methods used, the set-backs that occur and the improvements made should be recorded in writing and, when practicable, on phonographs.

PALMER, Wichita, Kan.

ANTIHISTAMINIC DRUGS IN THE TREATMENT OF UPPER RESPIRATORY TRACT INFECTION. JOHN S. GORDON, Laryngoscope **57**:1265 (Dec.) 1948.

Admitting that it is difficult to ascertain the effect of treatment on self-limited disease, Jordan has endeavored to evaluate the effect of the antihistaminic drugs on infections of the upper respiratory tract. Tripelennamine hydrochloride (pyribenzamine hydrochloride®) was the most satisfactory and seemed to be beneficial in most cases. In a few cases the common cold may even have been aborted in six to eight hours.

HITSCHLER, Philadelphia.

DR. THOMAS WILLIS (1621-1675): A GREAT SEVENTEENTH CENTURY ENGLISH ANATOMIST AND CLINICIAN WHO ANTICIPATED MANY MODERN DISCOVERIES. WALTER A. WELLS, Laryngoscope **59**:287 (March) 1949.

Willis was a great anatomist, clinician and philosopher. His name is linked with the discovery of the accessory nerve (Willis' nerve), the circle of Willis, the phenomenon known as paracusis Willisii, or Willisiana, and with certain facts concerning diabetes (Willis' disease). He wrote on many other subjects, and his works were widely accepted and read at the time. Much of his writing showed a remarkable anticipation of the modern theories of medicine. Some medical historians have disparaged Willis and his works. He did have certain faults, which caused some antagonism, but was nevertheless one of medicine's great and should so be remembered.

HITSCHLER, Philadelphia.

THE ROLE OF THE UPPER RESPIRATORY MUCOUS MEMBRANE IN THE PATHOGENESIS OF POLIOMYELITIS. W. LLOYD AYCOCK, Laryngoscope **59**:335 (April) 1949.

Aycock reviews and analyzes the poliomyelitis-tonsillectomy problem concisely and clearly. In some of the surveys the relationship is diluted or lost because of statistical considerations and because of different points of view. For instance, one must follow hundreds of patients who have recently undergone tonsillectomy before one encounters a patient who has poliomyelitis, but if one examines many patients with bulbar poliomyelitis, one soon finds some on whom the operation has recently been performed.

The conclusion arrived at is: Don't do elective tonsillectomies when the virus of poliomyelitis is prevalent.

HITSCHLER, Philadelphia.

HEMATOLOGY. JOHN JOSEPH SHEA and JOHN JOSEPH SHEA JR., Laryngoscope **59**:693 (July) 1949.

Many of the recent advantages in hematology are of interest to the otolaryngologist. These are discussed with emphasis on the newer drugs and methods of treatment.

HITSCHLER, Philadelphia.

PSYCHOSOMATIC ASPECTS OF DISEASES OF THE EAR, NOSE AND THROAT. LESTER D. COLEMAN, Laryngoscope **59**:709 (July) 1949.

In this article Coleman shows with concrete examples how important is psychosomatic therapy and how disastrous are the results if such therapy is omitted when needed. He shows how a tonsillectomy should be handled to avoid psychic trauma. He also illustrates with other conditions, such as tinnitus, and deafness, cosmetic disfigurations and hysterical aphonia. The paper is clear, practical and helpful.

HITSCHLER, Philadelphia.

CAROTID PAIN. JEROME A. HILGER, Laryngoscope **59**:829 (Aug.) 1949.

Imbalance of the autonomic nervous system is exhibited perhaps principally in alterations in vasomotor control of the carotid arterial tree. Clinically, control of the arterioles rather than of the large arteries is the important phase. The wheal and edema of allergy are probably due to such an imbalance, and an allergic person is one with an unstable autonomic nervous system. Passive dilatation, with a resultant stretch in the arterial walls behind an angiospasm, results in pain in the affected area. The pain is a throbbing one, being accentuated by the pulse. Supportive pressure frequently gives some relief. A common site is at the bifurcation of the carotid artery, with a tender pulsating swelling present. Facial pain which does not correspond in area to distribution of the sensory fibers is usually of this nature.

Intranasal operation, by altering the sympathetic imbalance, may give relief from pain, but, unfortunately, this relief is only temporary. It is one of the reasons such surgical procedure has fallen into disrepute.

Autonomic imbalance may be inherited. Emotional changes may be physical, such as exposure to certain inhalants and food or changes in temperature, humidity and light. Treatment is directed at the cause, and, in addition, certain drugs which counteract the angiospasm, such as nicotinic acid, tripelennamine (pyribenzamine®), ergotamine tartrate and tetraethylammonium chloride, are useful. Case histories are given.

HITSCHLER, Philadelphia.

THE PATHOLOGY OF THE BRONCHUS. OSCAR AUERBACH, New York State J. Med. **49**: 900 (April 15) 1949.

The history of bronchial carcinoma is "mixed," but the tendency today is to consider that the neoplasm arises "exclusively from the bronchial epithelium." The types are squamous cell carcinoma, undifferentiated cell carcinoma and adenocarcinoma; the first two constitute the largest group. Carcinoma of a bronchus is found in about 1 per cent of all autopsies and in about 8 per cent of all other deaths from carcinoma. The condition is apparently on the increase; at least it is being observed more frequently. In this series of 50 autopsies, 92.5 per cent of patients were men, and 7.5 per cent were women; 15 per cent were Negroes. The greatest number were in the sixth and seventh decades of life. The right and left lungs were about equally involved; the upper lobe was involved more frequently than the lower. The author classifies carcinoma of the bronchus in three groups, the hilar, the pulmonary and the peripheral types, on the basis of "gross appearance." Microscopically, the lesions in nearly all cases were inclined toward metastasis. The adenocarcinomas often metastasized elsewhere than in the chest. Two tables and seventy references are set down for those who wish to study intensively the pathology of the lung.

VOORHEES, New York.

THE ROLE OF BRONCHOSCOPY IN DIAGNOSIS AND TREATMENT (SYMPOSIUM—PRIMARY CARCINOMA OF THE LUNG). LOUIS H. CLERF and PETER HERBUT, New York State J. Med. **49**:907 (April 15) 1949.

These authors report their studies from Jefferson Hospital, Philadelphia. It is frankly admitted that bronchoscopy does not solve all problems in carcinoma of the lung. Some failures are due to inability to reach the site with the bronchoscope; others occur because it is not possible to obtain a section from the area of greatest involvement. One may obtain infiltrated specimens only. Later, studies taken elsewhere may show up the failures, because conditions may then be more

favorable. During August 1947, a follow-up was attempted to compare cytologic diagnosis with other methods. All those instances of cancer diagnosed by cytology were confirmed by bronchoscopy or other procedures. In 161 of 180 cases (89.7 per cent), neoplastic cells were found in tissues removed bronchoscopically. A positive or inferential diagnosis was made by bronchoscopy in 113 cases (62.8 per cent), but cytologic studies were positive and confirmed in some 89.7 per cent, a difference of 27 per cent. In the cytologically positive cases, surgical extirpation was more frequently possible than in those in which diagnosis was bronchoscopic. The authors state: "It is our opinion that, when . . . roentgen studies . . . are made and when all patients with questionable shadows . . . are subjected to bronchoscopy so that secretions may be secured for cytologic examination, the diagnosis of bronchogenic carcinoma will be made earlier, . . . and the prognosis . . . will become more hopeful." VOORHEES, New York.

CARCINOMA CELLS IN SPUTUM AND BRONCHIAL SECRETIONS. L. B. WOOLNER and J. R. MCDONALD, Surg., Gynec. & Obst. **88**:273 (March) 1949.

Attention is directed to the variation in the appearance of carcinoma cells in sputum or bronchial secretions, depending on the histologic type of tumor in the bronchus. Cancer cells originating in a bronchogenic carcinoma of small cell (oat cell) type or in a keratinizing squamous cell type provide the most distinctive morphologic characteristics. A diagnosis of the histologic type of tumor present was usually possible in the case of these two cell types. In most other bronchogenic carcinomas, especially in the more undifferentiated tumors, a diagnosis of "carcinoma cells present" was made without reference to the histologic type.

Analysis of 150 cases in which the smears were diagnosed as positive for carcinoma cells was carried out. In 141 of the 146 cases a final diagnosis of primary or metastatic carcinoma of the lung was made. In 3 cases false positive diagnoses were made, and in 2 cases the final diagnosis was not definitely established.

Carcinoma cells in sputum or bronchial secretions provided the only preoperative microscopic evidence of cancer in 25 (43 per cent) of a total of 58 cases in this series in which surgical exploration was carried out. Of 24 cases of bronchogenic carcinoma in which the lesion could be removed, cancer cells in the smears provided the only microscopic evidence of cancer in 9 cases (37 per cent).

FRIEDBERG, Chicago.

GASTRIC RESECTION FOR ESOPHAGITIS AND STRICTURE OF ACID-PEPTIC ORIGIN. O. H. WANGENSTEEN and N. L. LEVEN, Surg., Gynec. & Obst. **88**:560 (May) 1949.

Relief from esophageal obstruction followed gastric resection in 6 patients who presented clinical evidence of esophagitis or esophageal stricture, or both. Three of the patients had in association duodenal ulcer; 1 had a gastric ulcer; 1, a congenitally short esophagus and repeated hematemesis of unknown origin, and 1 patient had a stricture of the lower part of the esophagus of unknown origin. Relief from the tendency toward ulcer by a satisfactory gastric resection seemed to be effective for the esophagitis and esophageal stricture resulting therefrom. The authors propose, as a corollary, that their observations constitute evidence that esophagitis may be a manifestation of ulcer. FRIEDBERG, Chicago.

UNCOMMON TUMORS OF THE SALIVARY GLANDS. M. C. WHEELOCK and T. J. MADDEN, Surg., Gynec. & Obst. **88**:776 (June) 1949.

Attention is called to the emphasis placed on mixed tumors of the salivary glands and their malignant phases, often to the neglect of other neoplasms arising in these areas. The authors present 13 uncommon tumors of the salivary glands, among them 2 adenomas, 2 hemangiomas, 1 lymphangioma, 1 perineural fibroma, 1 papillary adenocystoma lymphomatosum, 1 leiomyoma, 1 leiomyosarcoma and 3 lymphomas. The group of lymphomas included lymphosarcoma, malignant lymphocytoma and atypical Hodgkin's disease.

FRIEDBERG, Chicago.

CONGENITAL CYST OF THE FACE AND NECK. ALBERT JENTZER, Pract. oto-rhino-laryng. **11**:103, 1949.

After discussing the three types of congenital cysts, branchiogenic tonsillar, mucoid and dermoid, and their differential diagnosis, the author presents a case of a dermoid cyst of the neck with a fistula. He differentiated the diagnosis from that of other cysts and eliminated the possibility of fistula due either to tuberculosis or syphilis. He excised the cyst and followed the excision with an injection of 40 cc. of a mixture of formaldehyde solution, alcohol and glycerin, effecting a complete cure.

PERSKY, Philadelphia.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Oliver E. Von Alyea, M.D., *President*

Lawrence J. Lawson, M.D., *Secretary*

Monthly Meeting, Nov. 7, 1949

Physiology of Respiratory Resistance. JOHN S. GRAY (by invitation).

The sequence of physiopathologic changes in progressive obstruction of the upper respiratory passages is described as (*a*) impairment of ventilation capacity which causes dyspnea and (*b*) hypoventilation, i. e., a ventilation inadequate to the requirements for gas exchange, which causes asphyxial changes in the arterial blood, consisting predominantly of a rise in free carbon dioxide but also a fall in oxygen saturation and p_H. Each of these blood changes eventually constitutes a serious threat, and restoration and maintenance of the airway is the only way to correct all three. As hypoventilation progresses, a crisis is reached in which respiration weakens because of the inhibitory effects on respiration of severe asphyxial changes. A vicious cycle is thus set up which terminates abruptly in apnea.

Favorable Results in Bulbar Poliomyelitis Treated as a Problem in Respiratory Obstruction. THOMAS C. GALLOWAY and MARTIN H. SEIFERT (by invitation).

It has been assumed usually that in cases of bulbar poliomyelitis the grave effects were due to direct action of the virus itself. We have quite a different concept of it. Treatment under nearly ideal conditions based on that concept resulted in recovery from bulbar poliomyelitis in 15 consecutive cases—all the cases of bulbar poliomyelitis among a total of 127 cases in nearly three years. This rate contrasts with a mortality rate in the same area for the same period of 24 per cent in cases of bulbar poliomyelitis and 3.4 per cent in all cases. (Our recent results, however, have not been so good with patients hospitalized late.)

Anoxia, with associated carbon dioxide excess, is frequently overlooked as an important cause of symptoms and serious pathologic changes. Especially is this true in secretional obstruction. We believe that the chief difficulty in severe bulbar poliomyelitis is usually interference with the swallowing mechanism, resulting in accumulation of secretion, food, fluid or vomitus in the airway. This interference results in anoxia, with its central and peripheral effects, and in secondary pulmonary changes. These are usually promptly relieved on clearing the airway, if done early.

In the 15 cases with recovery reported, the following measures had been used: (1) adequate postural drainage at 25 to 35 degrees; (2) continuous water suction; (3) oxygen and parenteral fluids and feeding; (4) use of the respirator when respiratory failure threatens, and (5) tracheotomy when indicated to prevent dangerous anoxia and accumulation of carbon dioxide; to prevent drowning in secretions; to permit by-passing of accumulation of fluid; to allow aspiration of the lower air passages, and to make safe the use of the respirator. Tracheotomy was necessary in 6 of the 15 cases.

The methods for the safeguarding of the airway must be effectively applied before there occur severe brain damage or irreversible changes in the pulmonary field, such as atelectasis, atypical pneumonia and pulmonary edema, which so often kill these patients. We believe that administration of carbon dioxide is not indicated in respiratory obstruction. This paper was published in full in *The Journal of the American Medical Association* (**141**:1, [Sept. 3] 1949).

DISCUSSION

DR. MARTIN H. SEIFERT: I have been seeing cases of poliomyelitis for about nineteen years, now, and for the past seven years have had charge of the poliomyelitis service at Evanston Hospital. We have had about 900 cases on the service, a large share of them during the last six or seven years. I was fortunate to have in the hospital a man with the courage and decisiveness of Dr. Galloway, because that is what it took and that is what it still takes.

This year, as Dr. Galloway told you, we have not had recovery in 100 per cent of the cases, but there are some extenuating circumstances. We have had 26 cases of bulbar poliomyelitis this year—about as vicious as anything I have seen. Five patients died. One patient came in about eight hours before death in a state of cyanotic flush, with elevated blood pressure and a dark flush. Tracheotomy was performed shortly after admission, and in spite of everything, several asphyctic crises developed, and she died. I think in general when one sees these spells of cyanosis or anoxia one can just about halve the expectancy of that patient's chance for recovery. We were not permitted to do a tracheotomy early on the second patient. The third patient, who came in about the fifth day of the disease, had already had several crises and had cyanotic pallor, in shock; tracheotomy was done, and about 150 cc. of fluid gushed from the tracheotomy wound. He died within three hours. In the fourth patient tracheotomy had apparently been done in time, but he died within a few hours. I cannot help feeling that in this case there had been enough virus infection to knock the patient out. The fifth patient was a pregnant woman who had been in bed because of hemorrhage. She had to have an emergency cesarean section following premature separation of a placenta previa. The next day she went into a state of crisis, and we put her in a respirator but finally had to perform a tracheotomy, and she did not survive.

Actually, the plea I would make is, first of all, for a change in attitude. I survived the first twelve years or so of my contact with poliomyelitis with an attitude that I am sure was not healthy: that bulbar poliomyelitis was a vicious fulminating infection of the brainstem and was fatal. That is a defeatist attitude. There is something that can be done to save the patient from it. As Dr. Galloway said, when one sees paradoxical breathing, it is amazing; this patient had marked paradoxical breathing, and when Dr. Galloway put a tube into the trachea the patient breathed normally. We physicians should change our attitude to one of hope and activity. We must include in the poliomyelitis team a laryngologist with courage and understanding. The treatment of poliomyelitis requires teamwork. The use of postural drainage and suction and oxygen is necessary. I would plead for the use of the respirator with cases of bulbar poliomyelitis, but unless one can keep a clear airway, it is dangerous. Wangensteen suction is of utmost importance to keep fluid and food out of the mouth: the patient may not be able to handle it if he vomits. After five or six days, if he begins to swallow, a tube is valuable.

I think we can tell a little sooner now when tracheotomy is indicated. When I see a patient come into the hospital, salivating and threshing about, I want a tracheotomy performed whether or not he is cyanotic, because I know cyanosis

will appear. Before the days of tracheotomy, I never saw a patient in such a condition for whom the ending was not bad.

A cyanotic flush is bad; cyanotic pallor is worse. I think we must stress the necessity of staying away from opiates and barbiturates. I think they are deadly. The best sedative I know is tracheotomy, oxygen and, sometimes, the respirator. We are not selling tracheotomy but the principle of keeping the airway open. It is true that in a place where only inexperienced help is available one will have to do more; no matter how experienced our own group is there are still cases with which we have trouble. We did 10 tracheotomies in 26 cases of bulbar poliomyelitis and 12 cases of respiratory obstruction this year.

Dr. Paul Holinger: Certainly, these papers are timely expositions of important work that has been done. The paper Dr. Gray presented summed up the problem better than anything I have heard. Dr. Galloway's work, of course, is known throughout the country and the world. I think tracheotomy is being accepted more than it used to be in tracheobronchitis, and gradually its use is being expanded to other fields. Tetanus, as Dr. Galloway said, has been an indication for tracheotomy at Cook County Hospital, and at Research Hospital we have advocated and used it with success in maintaining the airway. In frequently recurring cases of postoperative pulmonary collapse, especially following esophageal surgery, tracheotomy is being used. In cases of cerebral hemorrhage with inability to swallow, in which the patient has aspirated fluids, it should also be considered.

The increased acceptance of tracheotomy has been in part due to great improvement in postoperative care of the tracheotomized patient. The operation is no longer associated with high mortality. Steam rooms, electric suction pumps, chemotherapy and good nursing care have all combined almost to eliminate the problem of post-tracheotomy atelectasis and pneumonia. These complications formerly led physicians to postpone tracheotomy until the patient could hardly be expected to recover, and the operation was too frequently regarded as only a last minute gesture.

I should like to correct one impression Dr. Galloway seems to have with regard to our work with carbon dioxide as an expectorant and its theoretic use in cases of laryngotracheobronchitis. This work was done four or five years ago when we used various chemical agents to determine the effect of expectorants, oxygen and carbon dioxide on the tracheobronchial tree. We found different actions of these chemicals at various levels of the bronchial tree. We found that carbon dioxide liquefied secretions and that oxygen had a drying effect. We tried this in conditions such as postoperative collapse in which there was considerable secretion, and also in cases of tracheobronchitis, and confirmed the experimental studies of the various effects of these gases. This has been misinterpreted by some who thought we were advocating the use of carbon dioxide in dyspneic or even cyanotic infants who had tracheobronchitis. This is an error, and I take this opportunity to correct the statement.

Dr. Frederick Hiller: About four or five years ago I made a few comments to your society with regard to some of Dr. Galloway's cases of tracheotomy. At that time not so much could be said about the procedure, even though it appeared to have saved the life of one or another of the patients. Meanwhile you have heard Dr. Galloway discuss his impressive series of cases. I think there is no doubt that he has saved the lives of a number of patients who would have died without tracheotomy.

Neurologists are seldom asked to see such patients these days, although we are much interested in the problem. We diagnose bulbar poliomyelitis on the ground of paralytic medullary complications, and we see such patients becoming asphyxic,

whereby abnormal secretions and drowning in their own lungs often complicates the picture. From a neurologic angle I think it should be possible to make a diagnosis of a primary polioencephalitis on the basis of characteristic cranial nerve paralyses pointing to the medulla oblongata. In the older literature, between 1900 and 1910, one will find such cases described. The characteristic abnormal secretion, which is not only pharyngeal but also bronchial, has also been observed. Many years ago pathologists, in describing severe inflammatory lesions in the medulla oblongata, stated that, whereas the spinal form of poliomyelitis shows affinity of the inflammatory and destructive process for the ganglion cells in the anterior horn, the inflammatory lesions in the bulbar type are found to be more diffuse throughout the medulla. Lately, Dr. Magoun and Dr. McCarter have verified these observations made by Wickman and others forty years ago.

I think there is no doubt that there are cases of bulbar poliomyelitis in which respiratory difficulties leading to anoxia and hypercapnia cause a secondary circulatory damage to the brain. Whether the whole bulbar syndrome is anoxic by nature is another question. Patients who have true bulbar poliomyelitis are, in my opinion, endangered primarily by paralysis of the respiratory center, to which, of course, is added the effect of respiratory paralysis itself.

I should like to propose another idea that may explain part of the clinical picture. What is the difference between acute bulbar poliomyelitis and acute bulbar paralysis as we see it, e. g., in apoplectic bulbar paralysis or in cases of myasthenia gravis with severe involvement of the muscles of the pharynx and larynx? The outstanding difference seems to be this abnormal secretion in poliomyelitis, which is obviously of central origin. The hypersecretion, with which the patient cannot cope, makes bulbar poliomyelitis resemble other conditions when tracheotomy is used efficiently. The hypersecretion in which patients with bulbar poliomyelitis may drown is, in my opinion, of central origin, probably due to an inflammatory lesion about the salivatory nucleus.

We physicians know many instances in which anoxia of the brain manifests itself, e. g., in severe hemorrhages and also in various other circumstances. From a pathologic angle, patients with bulbar poliomyelitis fail to show the typical signs of anoxia and certain other manifestations which he did not describe, such as, for instance, loss of consciousness and convulsions, which are usually not seen in our patients. I think that hypercapnia may be more important a factor than anoxia, and, if I understand Dr. Galloway correctly, his thoughts are very similar.

Would it be of any advantage to give these patients alkalis, in the form of ammonia or otherwise, to counteract the acidosis?

Dr. John S. Gray: Dr. Hiller asked about the possibility of giving alkalis for acidosis. Of course it would be possible to give sodium carbonate, for example, not an ammonium compound. Giving alkalis will not reduce the high level of free carbon dioxide in the blood nor correct the anoxemia, although it might reduce the acidosis. I think to correct the latter condition without correcting the other would accomplish very little.

Dr. Thomas C. Galloway: We are firmly convinced that in the majority of cases bulbar poliomyelitis should be treated chiefly as a problem in secretional obstruction until some direct attack on the virus is possible.

I am glad Dr. Holinger agrees that the administration of carbon dioxide is not indicated in respiratory obstruction. Tracheotomy, of course, is not a dangerous thing, as Dr. Holinger pointed out, and has many widening uses. On occasion, laryngologists stand more in need of education than do general practitioners about the necessity and safety of this measure.

Dr. Hiller has brought up the question of pathologic and clinical correlation. It is a long story and the answer is not easy. From an extensive experience with monkeys and human beings, Dr. Bodian of Johns Hopkins says the attempt can easily lead to serious error. It must be remembered that nearly all of the patients with poliomyelitis who come to the autopsy table have died of asphyxia. It is hard to separate virus from asphyxial effects on the central nervous system which may markedly increase secretions. We do use the bronchoscope, but in a moribund patient who is resistant it may add too much strain. I have come back to believing that it is often best to do a tracheotomy immediately rather than attempt to insert a bronchoscope. The bronchoscope does help to prevent emphysema.

Heating of the Human Maxillary Sinus by Microwaves. JOHN J. BALLENGER and STAFFORD L. OSBORNE (by invitation).

This paper will be published in a future issue of the ARCHIVES.

DISCUSSION

DR. STAFFORD L. OSBORNE: The only known factor that is really definitely proved is that high frequency current will and does produce heat in living tissue. How much it does produce is always a question. Various methods have been used to see whether or not the temperature can definitely be raised. Our work has shown some slight rise in temperature, not very pronounced but still statistically significant on a 5 per cent level of probability. One wonders whether such a slight elevation might have any clinical value. I think we physicians forget that the heat capacity of the tissues is high and that they will absorb tremendous amounts of heat without showing a change in the basic level. It is quite conceivable to me that we might get excellent clinical results without a large increase in temperature. Increased temperature does increase blood flow. I am a little more concerned with the propaganda we shall inevitably find as a result of this new form of radiation. It should not be associated with the term "radar."

With the use of conventional diathermy radiation was measured in terms of 50 to 750 meters in wavelength. This was succeeded by short wave diathermy with a wavelength of 24 to 6 meters, which is now meeting competition with this new form of radiation. Most of us knew experimental work was being done with wavelengths of 1 meter, but believed this was merely experimental laboratory work and would never have clinical application. With the advent of World War II, the magnetron tube was perfected, giving a wavelength between 12 and 13 cm. Short wave diathermy has approximately a frequency of 12 million to 100 million cycles per second. We have now jumped the development of the microtherm to 3,500,000,000 cycles per second. We have never been satisfied to assume that the only effect of diathermy would be heat. The German literature has been full of other effects than heat. It was stated that certain organisms could be definitely killed with a definite wavelength, while others would be stimulated to further activity. Later, evidence was shown that most of the claimed effects were the results of uncontrolled research and could not be substantiated. A paper coming from an excellent center showed that when one used microwave diathermy blood flow was materially increased, but that when short wave diathermy was used the blood flow was not increased, or if there was any change it was backward. That work was duplicated in two or three laboratories, and these results could not be duplicated.

The matter of radiation affecting the eyes is creating a good deal of attention. Having done some work in short wave diathermy, I thought it would be a good idea to duplicate this work again, and we have found no ill effects when using

microwaves. Since then more extensive work has shown lenticular opacities to be present when microwave radiation is used. The thing I am afraid most people overlook is that the temperature secured in these studies is beyond the temperature that should be used in practice. I do think there is not anything new about heat producing lens opacities, but I do believe the danger is not great if one uses caution. Diathermy is not innocuous. It is dangerous in untrained hands, either short wave or the newer method. A good deal more work will have to be done before its rightful place can be determined. But one point is the small size of the directors used, which greatly limits the area to be heated. For the otolaryngologic and ophthalmologic specialty that would be an advantage, because with short wave it is difficult to get electrodes small enough. Another important factor is the matter of dosage. No one yet has been able to state what constitutes an accurate dose. There is no way of determining dosage other than by tissue tolerance. In your specialty, I hope you physicians will not be misled by articles coming out that state a certain output from the generator is used. You are told to set a machine at a given setting, but there is no guarantee that two patients are going to absorb the same amount of energy, although the output offered is identical, and that should be clearly kept in mind. Energy absorption determines the tissue temperature. That may explain some of the difficulty one gets into in determining the value of this work. There is no assurance that all the patients received the same amount of heat. I would suggest to anyone wishing to duplicate the work that he consider the use of thermistors instead of thermocouples for taking measurements. At the present time, one has to take the couples out and, after heating, reinsert them. In this way a great deal of heat is lost, and the highest temperature is probably not being attained.

Book Reviews

Blakiston's New Gould Medical Dictionary. First edition. Edited by Harold Wellington Jones, M.D.; Normand L. Hoerr, M.D., and Arthur Osol, Ph.D. With the cooperation of an editorial board and eighty contributors. Price, $8.50 (textbook edition). Pp. 1294, with 252 illustrations on 45 plates, 129 in color. The Blakiston Company, 1012 Walnut St., Philadelphia 5, 1949.

The Blakiston Company describes this work as "a modern, comprehensive dictionary of the terms used in all branches of medicine and allied sciences, including medical physics and chemistry, dentistry, pharmacy, nursing, veterinary medicine, biology and botany, as well as medicolegal terms, with illustrations and tables." This description is a concise summary of what is contained in this very comprehensive volume. Every physician needs a dictionary, and the Blakistons have been publishing medical dictionaries under the title of "Gould Medical Dictionary" since 1890, all of them having been accepted as standard. This present work, however, is in no sense a revision of any of the excellent dictionaries of the past, although based on them, and the new terms have multiplied greatly. It is a completely new reference work, compiled by a staff of experts, and it is now many years since a really new work of this character has been put forth.

The format and printing are excellent; the many illustrations are very well done, and the binding is semiflexible, making a very compact and easily handled volume. Needless to say, the reviewer could only "spot check" the contents, but in doing this he was unable to find any mising term or inadequate description. It appears to be the most complete and thorough medical dictionary ever published, and as such it should be in every medical library, private or institutional, especially in view of the fact that no new editions have appeared in recent years.

The 1949 Year Book of the Eye, Ear, Nose and Throat. Edited by Derrick Vail, M.D., D.Oph. (Oxon.), Professor and Director, Department of Ophthalmology, Northwestern University Medical School, and Samuel J. Crowe, M.D., Professor of Otology, Rhinology and Laryngology, Johns Hopkins University. With the collaboration of Elmer W. Hagens, M.D. Cloth. Price, $5. Pp. 580, with 125 illustrations. The Year Book Publishers, Inc., 200 E. Illinois St., Chicago, 1950.

In this issue of the "Year Book of the Eye, Ear, Nose and Throat," Dr. Vail competently carries on that portion of the work previously performed by Louis Bothman. He has been successful in bringing together those articles which seem to be the most valuable to the general practitioner and specialist in a concise form, in conformance with the policy laid down in previous editions. The second large portion of the book is adequately handled by the editors in their usual manner, the contents being in two large sections, on the ear, and on the nose and throat.

In both portions of the book, the comments of the editor are appropriate and noteworthy. The articles selected have been well chosen, and it is belived that this edition will enjoy the same amount of popularity as the previous ones.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. William H. Johnston, 1515 State St., Santa Barbara, Calif.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit 26, Mich.
Place: San Francisco. Time: June 28-30.

American Academy of Ophthalmology and Otolaryngology

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles 14.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 8-13, 1950.

American Broncho-Esophagological Association

President: Dr. Leroy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Hotel Mark Hopkins, San Francisco. Time: May 25-26, 1950.

American Laryngological Association

President: Dr. Ralph A. Fenton, 1020 S. W. Taylor St., Portland, Ore.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Hotel Mark Hopkins, San Francisco. Time: May 23-24, 1950.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Hotel Mark Hopkins, San Francisco. Time: May 25-27, 1950.

SECTIONS:

Eastern.—Chairman: Dr. Robert L. Moorhead, 125 Remsen St., Brooklyn, N. Y.
Southern.—Chairman: Dr. William D. Stimson, 899 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. J. Marion Sutherland, 3001 W. Grand Blvd., Detroit.
Western.—Chairman: Dr. J. B. Naftzger, 6777 Hollywood Blvd., Hollywood, Calif.

American Otological Society

President: Dr. Philip E. Meltzer, 20 Charlesgate, W., Boston.
Secretary: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 3, N. Y.
Place: Hotel Mark Hopkins, San Francisco. Time: May 21-22, 1950.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Samuel F. Kelley, 47 E. 61st St., New York 21.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. George E. Shambaugh Jr., 55 E. Washington St., Chicago.
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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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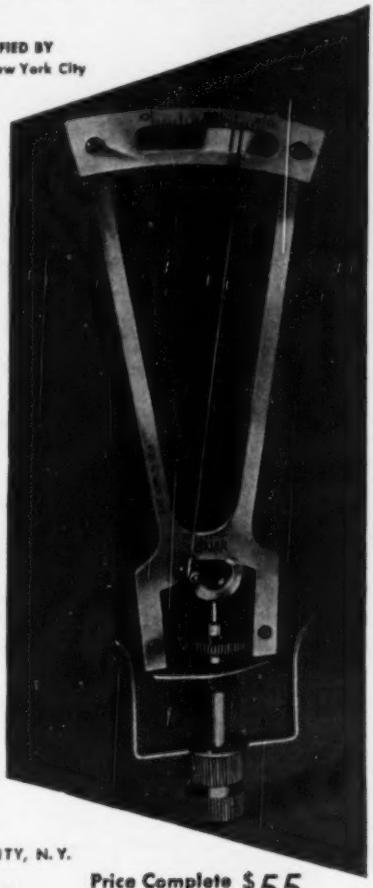

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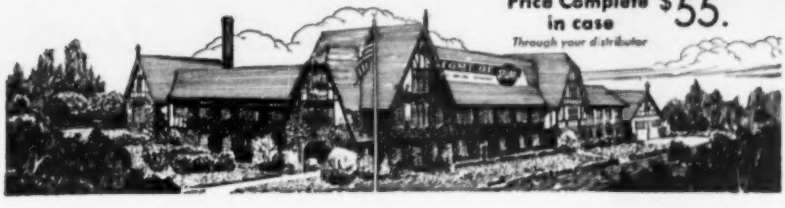

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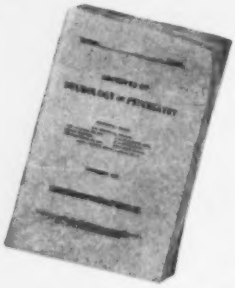

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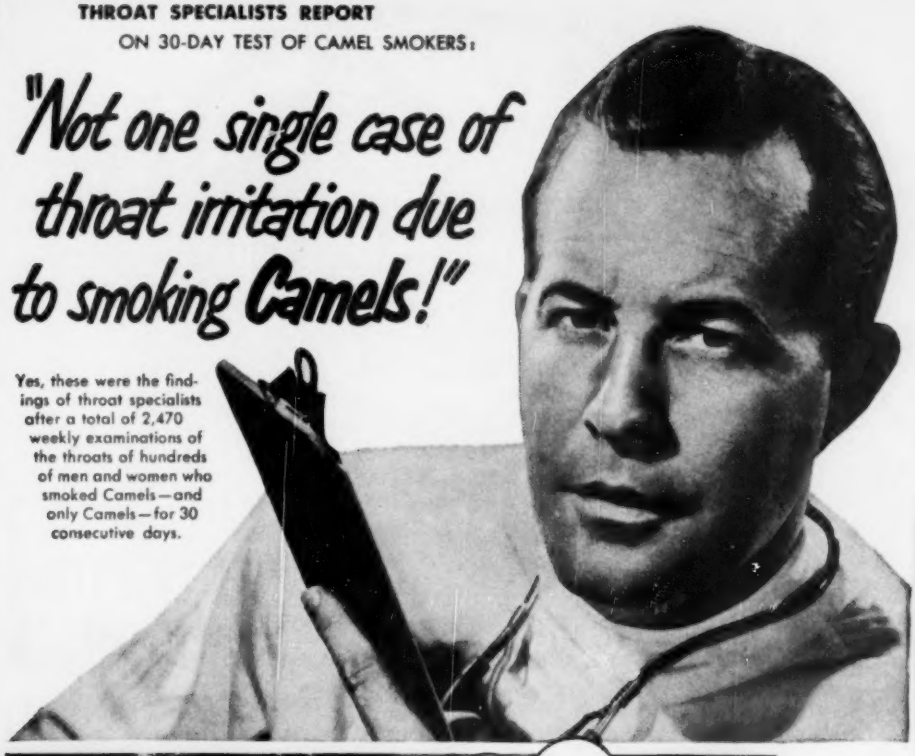

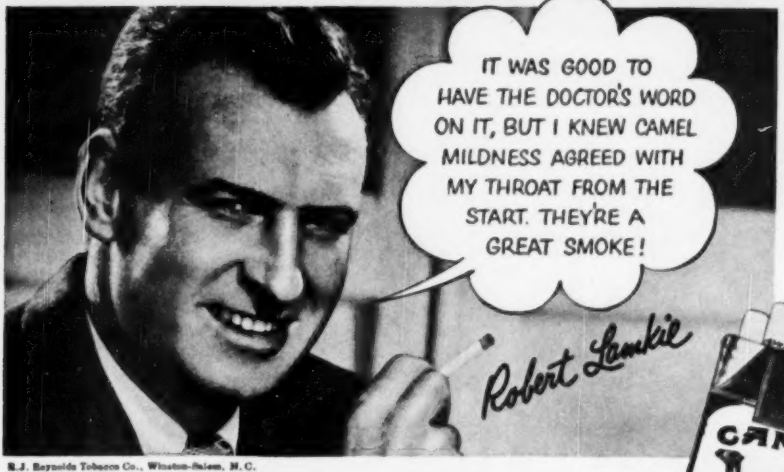
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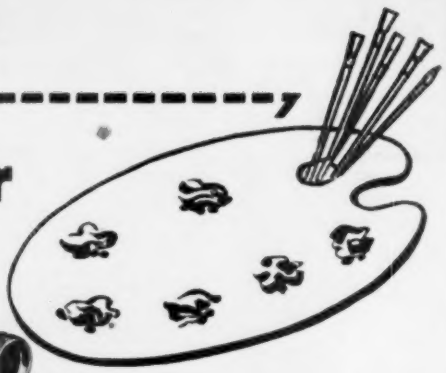

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